NERVE CONDUCTION STUDY AND GONIOMETRY IN HYPOTHYROID WOMEN

Dissertation submitted to

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY,

CHENNAI – 600032

In partial fulfillment of the requirement for the degree of

Doctor of Medicine in Physiology (Branch V)

M.D. (PHYSIOLOGY)

APRIL 2017

DEPARTMENT OF PHYSIOLOGY

COIMBATORE MEDICAL COLLEGE

COIMBATORE – 14

CERTIFICATE

This dissertation entitled **"NERVE CONDUCTION STUDY AND GONIOMETRY IN HYPOTHYROID WOMEN"** is submitted to The Tamil Nadu Dr. M.G. R Medical University, Chennai, in partial fulfillment of regulations for the award of M.D. Degree in Physiology in the examinations to be held during April 2017.

This dissertation is a record of fresh work done by the candidate **Dr. K.SYED MADHAR SHAH ,** during the course of the study (2014-2017).

This work was carried out by the candidate himself under my supervision.

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DECLARATION

I Dr. K. Syed Madhar shah solemnly declare that the dissertation entitled " NERVE CONDUCTION STUDY AND GONIOMETRY IN HYPOTHYROID WOMEN " was done by me at Coimbatore Medical College, during the period from July 2015 to June 2016. Under the guidance and supervision of **Dr.P.MURUGESAN. M.D.,** Professor, Department of Physiology, Coimbatore Medical College, Coimbatore. This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch - V) in Physiology.

I have not submitted this dissertation on any previous occasion to any University for the award of any degree.

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ACKNOWLEDGEMENT

ACKNOWLEDGEMENT

I express my sincere thanks to our respected Dean, **Dr.A.EDWIN JOE. MD, B.L.** Coimbatore Medical College, Coimbatore for permitting me to conduct the study.

I thank **Dr.MANI. M.D.,** Vice Principal, Coimbatore Medical College, Coimbatore for his encouragement and suggestions in completing the study.

I am extremely grateful to my beloved and respected Professor D**r. N. NEELAMBIKAI. M.D.,** Head of the Department of Physiology, Coimbatore Medical College, for her encouragement in helping me to take up this study. I express my heart - felt gratitude to her, for her moral support and encouragement throughout the conduct of the study and also during my post graduate course. I owe my sincere thanks to her.

I will ever remain in gratitude to **Dr.R.SHANMUGHAVADIVU M.D.,** Professor, Department of Physiology for her valuable support and guidance throughout my study.

I sincerely thank **Dr. P. MURUGESAN M.D.,** Professor, Department of Physiology for his valuable suggestion and

encouragement throughout the period of my study. I express my gratitude to him for his valuable time and patience that helped me to complete this study under his expert guidance

I am highly obliged to **Dr. D. SELVAM M.D.,** Associate Professor, Department of Physiology, for his motivation to perform this work.

My sincere thanks to beloved teachers **Dr.A.Moorthy M.D., Dr. S. Kavitha M.D., Dr.E.S. Manikandan M.D., Dr.S.Subhashini M.D., Mrs .D. Revathy Msc. , Dr. Abbas M.D., Dr. Thenmozhi M.D, Dr.Angel Deepa M.D,** Assistant Professors, Department of Physiology for their valuable opinion and help to complete this study.

I would like to thank all my **tutors** for their support in completing this study.

I sincerely thank **Dr. Sujatha**.**M.D;** Assistant Professor of Community Medicine for her timely help and guidance in doing the Statistical work.

I sincerely thank Mr. Pradeep, Neurology Technician for helping me in performing the Nerve conduction study.

I profusely thank the Medicine Department, Neurology Department and Biochemistry Department , Community Medicine

Department of Coimbatore Medical College and Hospital, Coimbatore for their cooperation and support.

I would grossly fail in my duty, if I do not mention here of my **subjects** who have undergone the pain and discomfort of the investigations during this study.

My sincere thanks to all my **fellow postgraduates** for their involvement in helping me in this work.

My **family and friends** have stood by me, during my times of need. Their help and supports have been of immense value for me to complete my study.

Above all I thank the **Lord Almighty** for His kindness and His blessings in every moment in my life.

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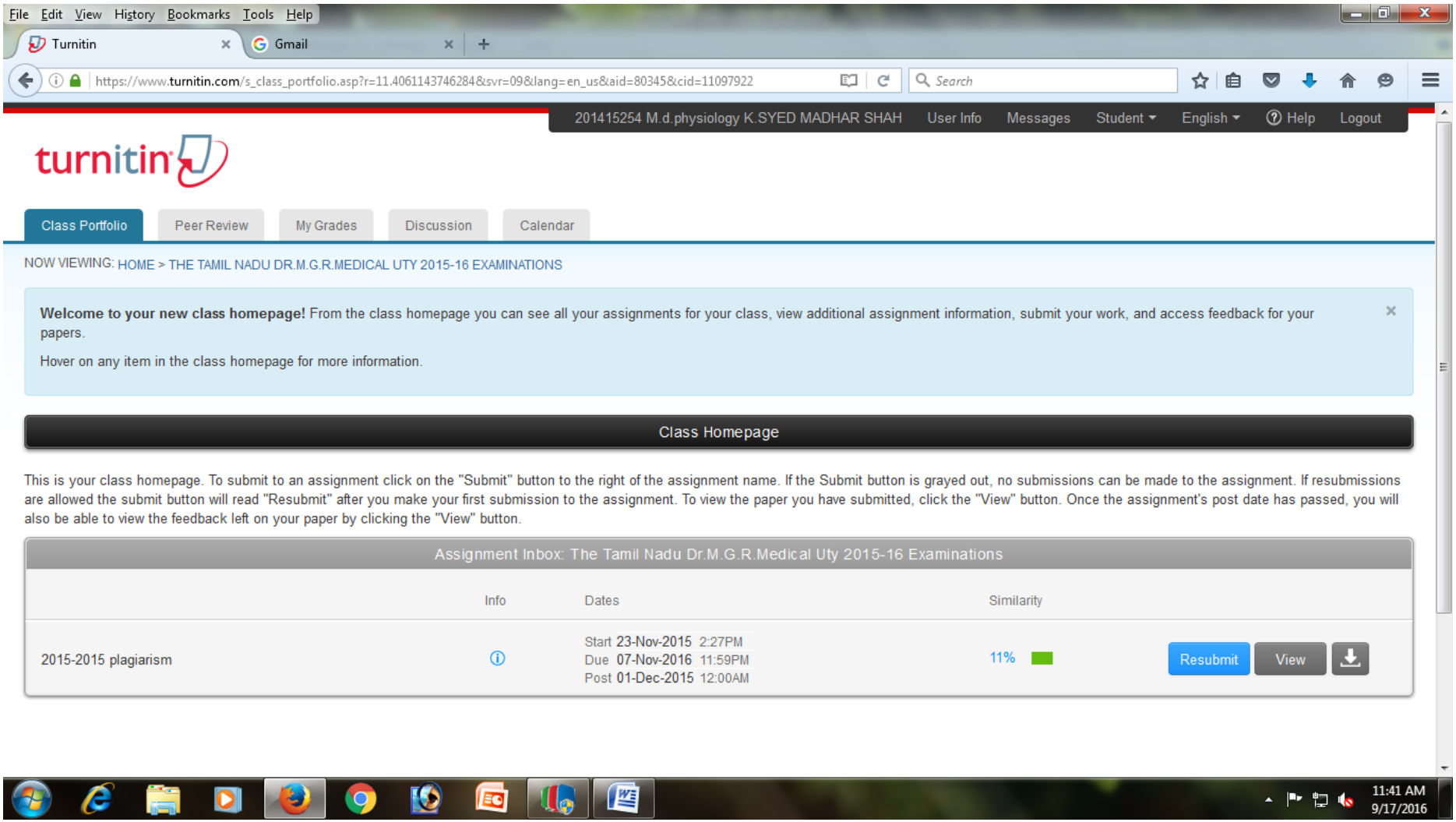
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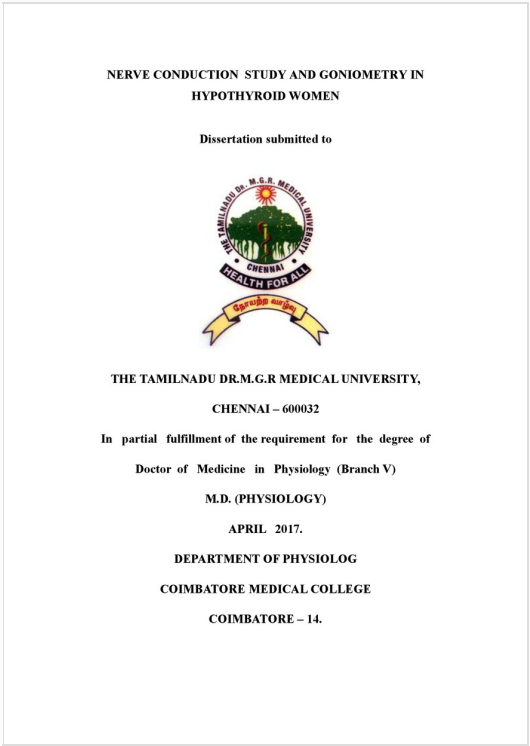

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CONTENTS

CONTENT

ABBREVIATIONS USED IN THE STUDY

NCS	Nerve Conduction Study
BMI	Body Mass Index
BMR	Basal Metabolic Rate
ECF	Extra Cellular Fluid
ECG	Electro Cardio Gram
ELISA	Enzyme Linked Immuno Sorbent Assay
Ms	Milli second
M/s	Meter / second
Mv	Milli volt
DIT	Diiodotyrosine
MIT	Monoiodotyrosine
TSH	Thyroid Stimulating Hormone
T3	Tri iodo thyronine
T4	Thyroxine or Tetra iodo thyronine
SCH	Sub Clinical Hypothyroidism
SNCV	Sensory Nerve Conduction Velocity
MNCV	Motor Nerve Conduction Velocity
Na / K ATPase	Sodium Potassium Adenosine Tri Phosphatase enzyme pump

IDD	Iodine Deficiency Disorder
ROM	Range of Movement
OPD	Out Patient Department
RIA	Radio immuno Assay
THs	Thyroid Hormones
TBG	Thyroxine Binding Globulin
µ	Microns or Micrometer

INTRODUCTION

INTRODUCTION

NAME OF THE STUDY

NERVE CONDUCTION STUDY AND GONIOMETRY IN HYPOTHYROID WOMEN

Hypothyroidism is the most common pathological hormone deficiency (1) as per Indian Thyroid Society (ITS). Around 42 million people in India suffer from diseases related to thyroid gland. Hypothyroidism being the most prevalent disorder affecting one in every eight women. Women are 5–8 times more susceptible to the disease (2).

Thyroid hormone is highly essential to lead a normal healthy life, as thyroid hormone has profound effects on almost every system of human body . Thyroid hormones regulate protein synthesis by affecting gene transcription and mRNA stabilization (3). It is essential for adequate and full development of fetal brain and the musculoskeletal system. In adults it has its effects on metabolism of carbohydrates, proteins, and fats. It is important in Red Cell production and its maturation .

Hypothyroidism has profound effects on the health of an individual . Hypothyroidism leads to infertility, loss of libido, leads to

hypertension, peripheral neuropathy affecting motor, sensory and mixed nerves producing chronic disability, derangements in metabolic functions, raised lipid profile, lethargy, increased weight gain, mental sluggishness and reduced nerve conduction velocity in adults.

The WHO estimates that about 2 billion people worldwide are iodine deficient , based on urinary excretion data (4). In India Iodine deficiency disorders account for 27 per 1000 . One in every eight women during their life time has risk for thyroid disorder (5).

Identification (diagnosis) and appropriate treatment of hypothyroidism in its early stages prevents its complications. Untreated hypothyroid patients may have preclinical asymptomatic small - fiber sensory neuropathy [6]. Clinical examination, Hormonal assay (TSH, T3, T4), nerve conduction studies all help in identification and improvement shown after the appropriate therapy and thus prevent further complications . Peripheral nerve involvement begins at the early phase of the disease – at the time of diagnosis.

PHYSIOLOGICAL ANATOMY OF THE THYROID GLAND

The thyroid is an endocrine gland situated in the anterior aspect of neck , in front of the larynx and trachea at the level of 5^{th}, 6^{th}, 7^{th} cervical and 1^{st} thoracic vertebrae behind, and at the level of the thyroid cartilage in front (7). It is made up of two lateral lobes connected by an isthmus . The average weight of the gland is 15 – 30 grams . (about 0.4 g/ kg body weight). It is larger in females especially during pregnancy and lactation. It is slightly larger in winter months .

Thyroid gland has rich blood supply from the superior and inferior thyroid arteries. It is one of the most highly vascular organs in the body with a blood flow of 4-6 ml / g / minute, which is further increased during hyperactivity .

Histologically the gland is made up of numerous spherical or oval vesicles (about 3 million) or follicles (acini) of 100 - 500 microns in diameter. These are lined by a single layer of cubical epithelial cells, which become taller as their metabolic activity increases (8). They are filled with a semifluid proteinaceous material called the colloid.

Thyroid gland develops as an invagination from the floor of the embryonic pharynx and grows downwards to form the isthmus and parts of the lateral lobes. Thyroglossal duct which connects the gland to the pharynx disappears early in the development .

The follicular cells synthesize two principal iodine containing hormones. They are Thyroxine and Triiodothyronine . Thyroxine is otherwise known as T4 and triiodothyronine is also known as T3. The daily secretion of T4 is about 80 micrograms. And daily secretion of T3 is about 4 micrograms. Apart from follicular cells, there are parafollicular cells which produce yet another hormone known as Calcitonin (Thyrocalcitonin) .

The systemic actions of T3, T4 on our body is widespread and obvious. These hormones have effects on almost all the systems in our body. Receptors for thyroid hormone are localized in nuclei of glial cells and neurons in different brain areas (9).

THYROID FUNCTION TESTS (11)

A. Based on Metabolic Effects of T4

1. Basal Metabolic Rate
2. Blood Sugar
3. Serum Cholesterol
4. Serum Creatinine

B. Based on Handling of Iodine

1. Protein Bound Iodine (PBI)
2. Butanol Extractable Iodine (BEI)
3. Radioactive Iodine Uptake

C. Other Investigations

1. Radiography
1. 2.Indirect Laryngoscopy
2. 3.Biopsy and Fine Aspiration And Cytology
3. Urinary Calcium Loss

DISORDERS OF THYROID HORMONE SECRETION

Hyposecretion of the thyroid hormone (THs) is known as Hypothyroidism in adults and Cretinism in infants. Hyper secretion of thyroid hormone (THs) is known as Hyperthyroidism.

NERVE CONDUCTION STUDY

Nerve conduction study (N C S) is part of the electro diagnostic procedures that help in establishing the type and degree of abnormalities of the nerves. N C S establishes diagnosis very early and more accurately than other electro diagnostic techniques because of its sensitivity in detecting conduction slowing (or block) which is an early indicator of nerve entrapment or peripheral neuropathy.

GONIOMETER :

The term goniometry is derived from two Greek words, gonia meaning angle and metron, meaning measure . Thus a goniometer an

instrument used to measure the angles. Within the field of physical therapy, goniometry is used to measure the total amount of motion at a specific joint.

Goniometry can be used to measure both active and passive range of motion. Goniometers are produced in a variety of sizes and shapes and are usually constructed of either plastic or metal. The common types of instruments used to measure joint are the bubble inclinometer and the traditional goniometer (10).

AIM & OBJECTIVES

AIM OF THE STUDY

The aim was to find out peripheral nerve conduction velocity, range and degree of movement of joints in the newly diagnosed hypothyroid women and comparing with the normal euthyroid women.

OBJECTIVES OF THE STUDY :

1. Comparison of motor nerve conduction velocity , range and degree of movement to electrical stimulation of nerves between normal euthyroid women and hypothyroid women.
2. Comparison of sensory nerve conduction velocity , range and degree of movement to electrical stimulation of the nerves between normal euthyroid women and hypothyroid women.
3. Analysis of motor and sensory nerve conduction velocities, range and degree of movement to electrical stimulation of the nerves in normal euthyroid women.
4. Analysis of motor and sensory nerve conduction velocities, range and degree of movement to electrical stimulation of the nerves in hypothyroid women.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The word thyroid is derived from Greek word Thyreos, means "A Shield" (11). The thyroid gland , located immediately below the larynx on each side of and anterior to the trachea , is one of the largest of the endocrine glands normally weighing 15 to 20 grams in adults . The thyroid secretes two major hormones, thyroxine and triiodothyronine, commonly called T4 and T3 respectively . Both of these hormones profoundly increase the metabolic rate of the body. Both these hormones maintain the level of metabolism in the tissues that is optimal for their normal function (12).

Complete lack of thyroid secretion usually causes the basal metabolic rate (BMR) to fall 40 to 50 percent below normal, and extreme excesses of thyroid secretion can increase the basal metabolic rate to 60 to 100 percent above normal. Thyroid secretion is controlled primarily by thyroid - stimulating hormone (TSH) secreted by the anterior pituitary gland. The thyroid gland also secretes calcitonin, an important hormone for calcium metabolism (13).

HISTORY OF THYROID GLAND

Ancient paintings found in Egyptian civilization emphasize the relation between thyroid gland and the women (14). In 1600 BC the Chinese were using burnt sponge and seaweed for the treatment of goiter(15).

It was thought in olden days that the main function of thyroid gland is to lubricate the trachea (14).

In 150 AD, Galen, referred to burnt sponge –'spongia usta' for the treatment of goitre. In 650 AD , Sun Ssu – Mo used a combination of seaweed, dried powdered mollusk shells and chopped up thyroid gland for the treatment of goiters.

Ali – ibn – Abbas was the first to discuss surgery as a treatment method for goiters in 990 AD. Jurjani's "Treasure of Medicine'' in 1110 AD, first associated exophthalmos, later associated Grave's disease, with the goitre.

The first references to the thyroid gland in western medicine is found in 1656 . In the early 18th century "thyroid gland was thought to be a vascular shunt to divert blood flow from the brain" .

In 1475 Wang Hei anatomically described the thyroid gland. In 1656, Thomas Wharton named it the thyroid gland, meaning "Shield'' due to its shape.

In 1811, Paris discovered iodine in the burnt ashes of seaweed and the idea to prescribe to goiter patients was developed. Prout was the first to recommend iodine in the treatment of goiters.

In 1835, Robert James Graves, an Irish doctor published his accounts on the exophthalmic goiter. Earlier It was known in the European continent as Basedow's disease. Karl Adolph Basedow had described the entity independently in 1840. In Switzerland in the 1880 s, Theodor Kocher demonstrated that total thyroidectomy caused hypothyroidism. Kocher

performed over 2000 thyroidectomies . Kocher was the greatest surgeon of the era . He was awarded the Nobel Prize for Medicine in 1909.

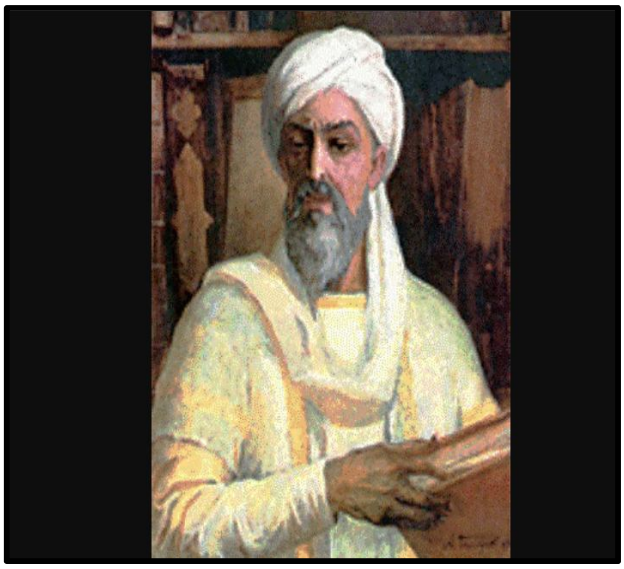
In 1880, Ludwig Rehn a German physician carried out the first thyroidectomy for exophthalmic goiter. In 1914, Edward Calvin Kendall isolated thyroxine, his crystalline extract had the correct structure and biological activity.

In 1883 at a meeting of the Clinical Society of London, Felix Semon suggested that the symptoms of Swiss patients who had a total thyroidectomy were very similar to English patients who had Myxedema.

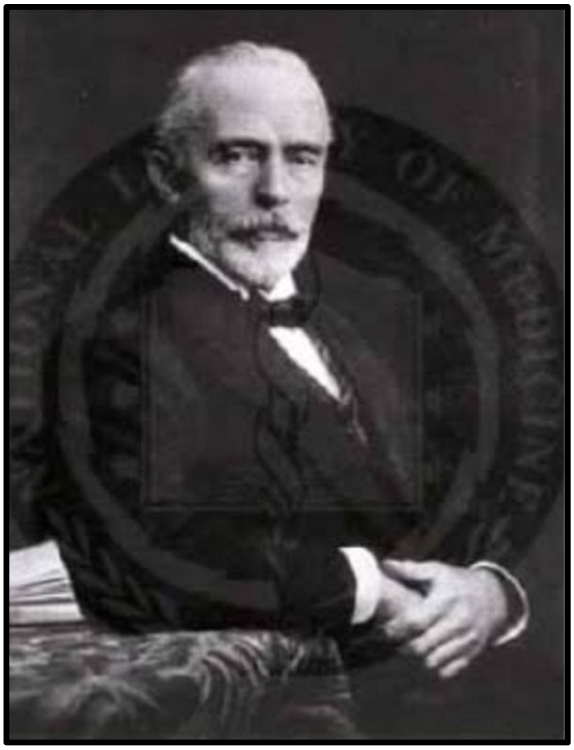
The synthesis of sodium L - thyroxine and its ability to be absorbed orally revolutionized thyroid replacement making it safe and cheap. In 1952 Rosalind Pitt – Rivers and her post-doctoral fellow Jack Gross discovered and synthesized tri-iodothyronine showing it was biologically more active than thyroxine (15) .

HISTORY OF NERVE CONDUCTION STUDY

History of neurophysiology is a combination of human intellect , perseveration and development of technology. The advances in clinical neurophysiology are closely related to the discovery of electricity. Details of few scientists who contributed to the development of clinical neurophysiology is given below :



Ali-ibn-Abbas



Emil Theodor Kocher 1841 – 1917

Rosalind Pitt- River

In 1791 Luigi Galvani the professor of anatomy at the University of Bologna discovered that the nerves were a good conductor of electricity and also believed that the oily envelope of the nerves rendered them good conductors of electricity. In 1851 DuBois Raymond recorded the action potential of voluntarily contracting muscle using jars of liquid as electrode which was the beginning of electromyography.

In 1850 Herman Von Helmholtz measured the conduction velocity of nerve in frog by mechanically recording the muscle twitch. Herman in 1870 stimulated the brachial plexus in axilla and recorded the muscle action potential from the surface of forearm . Measurement of motor conduction velocity employing muscle action potential rather than muscle twitch was carried out by Piper in 1909. Later, Harvey and Kutfer in 1944 applied nerve conduction studies in patients with peripheral neuropathy. Hodes Laravee and German in 1948 first calculated the conduction velocity by stimulating the nerve at different levels .

Recently , many neurophysiological tests such as F wave and blink reflex have emerged as clinically useful. Nerve conduction studies are sensitive, reliable, and objective measures of peripheral nervous system. They are sensitive enough to detect abnormalities even when the clinical examination is normal .

DEVELOPMENTAL CONSIDERATION OF THYROID GLAND

EMBRYOLOGY

Thyroid gland arises as a median outgrowth from the floor of the pharynx near the base of the tongue. The foramen caecum of the tongue indicates the site of origin and the thyroglossal duct marks the path of migration of the thyroid gland to its final adult location .

Ectopic location of thyroid gland :

There may also be functional thyroid gland

1. Associated with the tongue (a lingual thyroid)
2. Anywhere along the path of migration of the thyroid gland or
3 . Extending upward from the gland along the path of the thyroglossal duct (a pyramidal lobe) (16) .

PHYSIOLOGY OF THYROID GLAND FUNCTION

Thyroid gland secretes tri-iodo-thyronine (T3) and thyroxine (T4) hormones in response to stimulation by Thyroid Stimulating Hormone (TSH) (4).

THYROID HORMONE TRANSPORT AND METABOLISM AND ITS MECHANISM OF ACTION

T4 is secreted from the thyroid gland in about twenty fold excess over T3 . Both hormones are bound to plasma proteins - Thyroxine Binding Globulin (TBG), Transthyretin (TTR), and Albumin .

Circulating thyroid hormones enter cells by passive diffusion and via specific transporters such as the monocarboxylate 8 (MTC8) transporter. After entering cells, thyroid hormones act primarily through nuclear receptors.

IODINE METABOLISM

Iodine is an essential micronutrient. It is required for the synthesis of the thyroid hormones. Once within the gland, iodide rapidly moves to the apical surface of the epithelial cells. From there, it is transported in to the lumen of the follicles by Sodium dependent iodide / chloride transporter, named Pendrin. (17). Iodine is essential in minute amounts for the normal growth and development and well being of all humans . The adult human body contains about 50 mg of iodine , and the blood level is about 8 – 12 micrograms / dl. The minimum daily iodine intake that will maintain normal thyroid function is 150 microgram in adults.

In the developed countries the average dietary intake is approximately 500 micrograms. Out of 500 micrograms of iodine 120 micrograms is taken up by the thyroid gland, which is the principal organ

to use iodine. Hence thyroid gland utilizes 80 micrograms of iodine for the synthesis of T3 and T4 (Thyroid Hormones), the remaining 40 micrograms diffuses back into the Extra Cellular Fluid (ECF). The circulating T3 and T4 are metabolized in the liver and other tissues, with the release of further 60 micrograms of iodine daily into the extra cellular fluid . Some thyroid hormone derivatives are excreted in the bile and some of the iodine in them is reabsorbed (enterohepatic circulation).

20 micrograms of iodine is excreted in stools daily and the remaining 480 micrograms is excreted in the urine daily . Thus the iodine homeostasis is maintained in our body (18). Thyroxin (T4), and Triiodothyronine (T3) contains 4 and 3 atoms of iodine respectively .

SOURCES

Sea foods are the best sources – example Sea fish sea salt, cod liver oil. Smaller amounts occur in other foods like milk, meat, vegetables, cereals, etc. The iodine content of fresh water is small and very much variable – about 1 - 50 micrograms.

GOITROGENS

Goitrogens are chemical substances leading to the development of goitre. They interfere with iodine utilization by the thyroid gland. The Brassica family of vegetables – cabbage, cauliflower may contain goitrogens. Most

important among the dietary goitrogens are probably cyanoglycosides and the thiocyanates .

The most obvious consequence of iodine deficiency is goitre, but there is a much wider spectrum of disorders. They include 1.Hypothryroidism 2. Retarded physical development and impaired mental function 3.Increased rate of spontaneous abortions and stillbirths 4 .Neurological cretinism including deaf mutism and 5. Myxedematous cretinism including dwarfism and severe mental retardation . (19).

In some persons with colloid goiter, the thyroid gland has an abnormality of the enzyme system required for formation of the thyroid hormones . Among the abnormalities, often encountered are the following :

1. Deficient iodide – trapping mechanism, in which iodine is not pumped adequately into the thyroid cells.
2. Deficient peroxidase system, in which the iodides are not oxidized to the iodine state .
3. Deficient coupling of iodinated tyrosine in the thyroglobulin molecule so that the final thyroid hormones cannot be formed.
4. Deficiency of the deiodinase enzyme , which prevents recovery of iodine from the iodinated tyrosine that are not coupled to form the thyroid hormones (this is about two thirds of the iodine), thus leading to iodine deficiency . (13).

GEOGRAPHICAL DISTRIBUTION OF THYROID PROBLEM

Goiter has ceased to be a major problem in many developed countries (although not eradicated) it continues to be a serious health problem in many third World countries . For example iodine deficiency is a health problem of considerable magnitude in India and the neighboring countries of Bangladesh, Bhutan, Myanmar, Indonesia, Nepal, Sri Lanka and Thailand. More people are affected and levels of severity are higher in South – East Asia than anywhere else in the World.

It has always been thought in India that goiter and cretinism were only found to a significant extent in the 'Himalaya goiter belt' which is the world 's biggest goiter belt.

It stretches from Kashmir to the Naga Hills in the east , extending about 2,400 km and affecting the northern States of Jammu and Kashmir Himachal Pradesh Punjab Haryana Delhi Uttar Pradesh Bihar West Bengal Sikkim Assam Arunachal Pradesh Nagaland Mizoram Meghalaya Tripura and Manipur.

In recent years renewed surveys outside the conventional goiter belt have identified endemic foci of iodine deficiency and the associated IDD in parts of Madhya Pradesh Gujarat Maharashtra Andhra Pradesh Kerala Karnataka and Tamil Nadu. In short, no state in India can be said to be entirely free from goiter.

The magnitude of the problem in India is far greater than what had been estimated in 1960s, when it was estimated that about 9 million persons were affected by goiter. Results of sample surveys conducted in 325 districts covering all the states / Union Territories have revealed that 263 districts covering all the prevalence of Iodine Deficiency Disorder is more than 10 percent.

It is estimated that more than 71 million people are suffering from goiter and other iodine deficiency disorders in the country .

GOITRE CONTROL

There are four essential components of National Goitre Control Programme. These are

1. Iodized salt or oil,
2. Monitoring and surveillance,
3. Manpower training, and
4. Mass communication.

IODIZED SALT

In India the level of iodination is fixed under the Prevention of Food Adulteration (PFA) Act and is not less than 30 ppm at the production point , and not less than 15 ppm of iodine at the consumer level.

IODIZED OIL

National institute of Nutrition, Hyderabad have successfully developed - Intramuscular injection of iodized oil (mostly Poppy – seed oil), iodized oil in safflower or safola oil.

ACTIONS OF THYROID HORMONE

The actions of T3, T4 on our body is widespread and obvious. These hormones have effects on almost all the systems of the body. The actions of these hormones on different systems is mentioned below.

1. GROWTH : Thyroid hormones are essential for normal growth and maturation of most of the tissues of the body . They are necessary for skeletal growth and maturation, ossification of cartilage, normal contours of the face, formation and eruption of teeth and for normal proportions of the body.
2. BRAIN AND NERVOUS SYSTEM GROWTH : Thyroid hormone is essential for the development of the central nervous system and must be present in adequate amount at the time of birth and during the first year of life . The action of thyroid hormones (THs) in the brain is strictly regulated, since these hormones play a crucial role in the development and physiological functioning of the central nervous system (CNS) (20)(22). Transient reductions in thyroid

hormone during critical periods of brain development can have devastating and irreversible effects on neurological function (23).

3. GENERAL METABOLISM AND CALORIGENESIS **(Heat Production)** : Thyroid hormones are important regulators of cellular oxidative mechanisms and maintain the metabolism of the tissues at a level optimal for the normal function . On the carbohydrate metabolism , it increases absorption of glucose (hexose) from the small intestines and thus helps in hyperglycemia . On the lipid metabolism , thyroid hormones favor synthesis of cholesterol . On the protein metabolism, thyroxine promotes protein anabolism, increasing protein synthesis and nitrogen retention, resulting in positive Nitrogen balance.

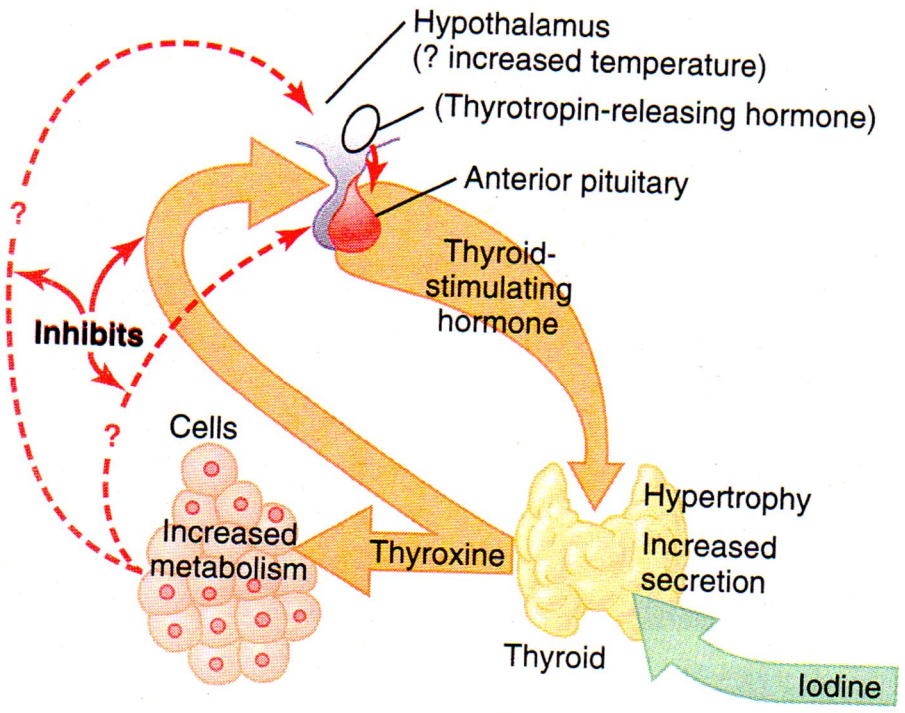
   Thyroid hormones have effects on bones, water, salts and vitamins and mineral metabolism .

4. EFFECT ON HEART : Thyroid hormones increase heart rate, force of contraction, cardiac output, systolic blood pressure and pulse pressure. It increases number of beta adrenergic receptors in the heart .
5. SKELETAL MUSCLE : For efficient muscle action, optimum thyroxine levels are necessary.
6. REPRODUCTIVE SYSTEM : Optimal amount of thyroxine is necessary for normal gonadal function.

7. LACTATION : Thyroid hormones are necessary for adequate lactation.
8. RELATION TO CATECHOLAMINES : Thyroid hormones have a permissive action on the calorigenic effect of adrenaline.
9. OTHER EFFECTS : Respiratory rate and depth may be increased due to increased metabolism . Gastro intestinal secretion, motility, appetite may be increased. Thyroid hormones are necessary for RBC formation.
10. THYROID ACTIVITY IN FETUS : Thyroid hormones exert multiple effects on neural development and function (24). The thyroid gland is active in fetus . Since the maternal TSH does not cross the placenta, the fetus is dependent on its own TSH, which appears in the pituitary at about 11^{th} week of intrauterine life. At birth TSH secretion and thyroxine levels increase (25).

Disruption of thyroid hormone production during fetal and early neonatal development leads to a suite of permanent deficits in intelligence and sensorimotor function in humans (20)(26).

Brain - derived neurotropic factor (BDNF) is a neurotropin critical for many developmental and physiological aspects of CNS function. Severe hypothyroidism in the early neonatal period results in developmental and cognitive impairments and reductions in mRNA and protein expression of BDNF in a number of brain regions(20)(27).



Regulation of Thyroid hormone secretion (28)

REGULATION OF THYROID HORMONE SECRETION :

Thyrotrophic or thyroid stimulating hormone (TSH) of anterior pituitary is the primary regulator of thyroid function. TSH is essential for the normal structural development and secretory activity of the thyroid gland.

HYPOTHALAMUS

↓+TRH

ANT. PITUTARY GLAND

↓+TSH

THYROID GLAND

↓

T3 & T4 (Bound and free form) .

(+ = stimulation . TRH = TSH Releasing Hormone . TSH = Thyroid Stimulating Hormone . ANT = Anterior (81).

HYPOTHYROIDISM AND NEUROPATHY :

Deficiency of thyroid hormone leads to hypothyroidism. Hypothyroidism is a chronic disease affecting a wide variety of systems such as excretory, digestive, cardiac and nervous system (29)(30). Hypothyroidism is more commonly associated with myopathy (proximal muscle weakness) (31), mononeuropathy, and sensorimotor axonal polyneuropathy (32), most typically carpal tunnel syndrome. Rarely a

generalized sensory polyneuropathy characterized by painful parasthesias and numbness in both the legs and hands can occur. In hypothyroidism the muscle contraction and relaxation are slowed down while duration is prolonged.

In hypothyroidism patients develop the usual manifestations of peripheral neuropathy like loss of reflexes, weakness of proximal muscle paraesthesia, decrease sensations for example vibration, joint - position and touch – pressure.

In patients with clinically overt and undiagnosed hypothyroidism, peripheral nerves dysfunction may be the main manifestation with which patients can present to the Out Patient Department (33). Neuromuscular ocular dysfunction in hypothyroidism includes ptosis, ophthalmoplegia, cranial nerve dysfunction and cosmetic changes (34)(32).

Treatment is correction of hypothyroidism with L – thyroxine.

Thyroid hormone action on brain development is essentially exerted through regulation of the expression rate of a number of genes some of which have been identified in the past 10 years (35). Receptors for thyroid hormone are localized in nuclei of glial cells and neurons in different brain areas (36).

DISORDERS OF THYROID :

Hypo function of thyroid gland in **infants** produces “CRETINISM”. It is due to lack of thyroid hormones at birth. The child may be

apparently normal at birth, but effects appear within weeks . The milestones in the child's development appear much later than normal , due to late myelination of nerve fibers. The stature is stunted (dwarfism). The limbs appear to be short and thick . The child is unattractive and ugly. The mental development is greatly retarded and child becomes an idiot. Pubertal sexual development is arrested. Basal Metabolic Rate is reduced , body temperature is subnormal, serum cholesterol is high and circulating thyroid hormone levels are low.

Early detection becomes difficult because the symptoms are nonspecific . Screening tests such as RIA of serum TSH in the first week after birth is useful.

Hypothyroidism has numerous effects on brain. It produces a hypo metabolic state. Hypo metabolic state following hypothermia is known to protect tissues from ischemic injury. A study provided evidence that hypothyroidism made neuronal tissue less vulnerable to severe ischemic insult (37).

Since the thyroid hormones dramatically affect the maturation of specific neuronal populations, the absence of these hormones during the period of active neurogenesis leads to irreversible mental retardation and is accompanied by multiple morphological alterations in the brain (38).

Transient reductions in thyroid hormone during critical periods of brain development can have devastating and irreversible effects on neurological function (39).

Hypothyroidism in adults produces " MYXEDEMA ". The onset is very gradual. The features are :

1. The face is puffy and the eyelids are baggy. It is due to deposition of fluid containing mucoprotein. The edema does not pit on pressure and is called myxedema. The body weight is raised, the thyroid gland may be enlarged or atrophied.
2. Skin is dry, coarse, scaly and cold. The hair is coarse and there is excessive loss of hair from the outer third of eyebrows and head (40).
3. The Basal Metabolic Rate is low (-30 to -45) with subnormal body temperature and reduced tolerance to cold .
4. Mental activity is diminished , with slowness of thought and speech (slow cerebration) , nervous reactions are slowed. Memory is impaired , muscle tone is poor . Voice may be hoarse.
5. There will be apathy , lethargy , muscular weakness and fatigability and dull appearance, though patients may be pleasant and good natured. (41)(42).
6. The pulse is slow and blood pressure is low, cardiac output is reduced and circulation time slowed. Heart may be dilated, ECG shows low potential waves and the T wave may be inverted .

7. Anemia is present , serum cholesterol is high. Serum T3, T4 , PBI are low.
8. Sex function is depressed with decreased sex drive (loss of libido). In women there may be menorrhagia, irregular bleeding or amenorrhea.
9. Myxedema may be an autoimmune disease and patients may have anti thyroglobulin antibodies in the blood . (Hashimoto's thyroiditis).

SUBCLINICAL HYPOTHYROIDISM :

Subclinical hypothyroidism (SCH) is defined as a biochemical state characterized by an elevated serum TSH concentration with concomitant normal serum free thyroid hormone levels is a common disorder (43)(44). Biochemically , subclinical hypothyroidism has been reported to be associated with abnormalities in serum lipids (45)(46) endothelial dysfunction (47) accelerated atherosclerosis and coronary artery disease (48).

HYPERTHYROIDISM

In this condition there is hyperplasia and hypertrophy of the thyroid gland and increased hormone secretion. The gland may be slightly enlarged (small goiter).

RMS – EMG – EP MARK II

The features are :

1. Increase in Basal Metabolic Rate (BMR) by 50 % to 100 % , Oxygen consumption , Carbon di oxide output and pulmonary ventilation are increased.
2. Bilateral exophthalmos.
3. Heart rate increased (100 – 160 beats / minute).
4. Fine tremor of the outstretched hand.
5. Muscle weakness (thyrotoxic myopathy).
6. Sexual function may be affected.

NERVE CONDUCTION STUDY

Nerve conduction study (NCS) is part of the electro diagnostic procedures that help in establishing the type and degree of abnormalities of the nerves. NCS establishes diagnosis very early and more accurately than other electro diagnostic techniques because of its sensitivity in detecting conduction slowing (or block) which is an early indicator of nerve entrapment or peripheral neuropathy . The technique consists of an electrical stimulation of somatic nerves and the recording of the evoked potentials , either from the muscles or from the nerves themselves . Thus structural as well as functional changes in the nerves can be evaluated by these nerve conduction studies early and accurately in the course of the neural disease(49).

INDICATIONS FOR NERVE CONDUCTION STUDY

1. To localize the site or level of lesion – determining whether the injury involves the peripheral nerve , neuromuscular junction, plexus, nerve root, or anterior horn cell.
2. To distinguish whether the injury is due to axonal loss or demyelination.
3. To diagnose mononeuropathies such as median nerve (carpal tunnel syndrome) or ulnar nerve.
4. To diagnose more generalized peripheral neuropathies such as diabetes mellitus, or inflammatory neuropathies such as Guillain - Barre syndrome (50).

PRINCIPLE

Apply electrical shock at one point of the nerve and record the signal from another point . The complexity of NCS lies in the clinical application and interpretation of results . To interpret the result of nerve conduction studies one should know the anatomical course of the nerve the muscle supplied by the nerve the normal conduction velocity of the nerve, the physiological basis of the conduction of the impulse in the nerves, the pathophysiologic response of the nerve and muscle to disease and the biological electrical signal.

ANATOMICAL AND PHYSIOLOGICAL ASPECTS NERVE CONDUCTION

A basic knowledge of physiological anatomy of peripheral nervous system is necessary to understand its pathophysiology and principles of nerve conduction study. Peripheral nerves are composed of multiple fascicles, each fascicle is a bundle of nerve fibers . Peripheral nerves contain three sheaths of connective tissue, endoneurium, perineurium and epineurium from inside out (51). Endoneurium is the connective tissue sheath present within the fascicle . It contains mainly collagen which is arranged longitudinally parallel to the nerve fibers and few fibrocytes . Endoneurium is present between the surface membranes of Schwann cells in which the axons are embedded (51)(52).

Perineurium is the sheath that surrounds each fascicle. It contains flat polygonal cells that are bound by tight junctions to form a continuous membrane . Perineurium is responsible for blood nerve barrier. It forms diffusion barrier to regulate the intrafascicular milieu (53)(54). Perineurium also provides tensile strength and flexibility to the peripheral nerves (55). Epineurium is the outermost sheath that loosely binds the fascicles. It is made of collagen fibers and fat. The blood vessels and lymphatics are present in the epineurium. It continues with the duramater of the spinal root (57)(56).

Peripheral nerves contain both efferent and afferent fibers. Efferent fibers leave the spinal cord via the anterior roots and innervate the muscles.

Afferent fibers enter the spinal cord via posterior roots that convey sensory impulses to brain . The afferent and efferent peripheral nerve fibers have a central neuron that lies in dorsal root sensory ganglion or anterior horn of the spinal cord respectively from which the axon projects .

CLASSIFICATION OF NERVE FIBERS

Erlanger and Gasser divided mammalian nerve fibers into A , B , and C . The A group is further subdivided into α , β , γ and δ .

ANATOMICAL AND PHYSIOLOGICAL ASPECTS :

The conduction velocity of the nerve depends on the fiber diameter, degree of the myelination and the inter nodal distance .

As the axon increases in size myelin sheath becomes thicker and the inter nodal distance becomes longer. The conduction therefore becomes faster. The diameter of the nerve axons varies between 0.2 and 20 μ. The nerve fibers are classified as myelinated and unmyelinated. The myelinated axons are surrounded by Schwann cells, but there is no Schwann sheath in unmyelinated fibers. The junction between two Schwann cells is known as the node of Ranvier, where the axons remain uninsulated.

The inter nodal distance which is the distance between the two nodes of Ranvier, depends on the spacing of Schwann cells at the time of myelination during development. Proliferation of Schwann cells does not occur afterwards, but the inter nodal distance increases during the growth of the nerve. Thus, the myelinated fibers have an early and longer inter nodal distance, larger diameter and wider spacing at the nodes of Ranvier.

Nerve fibers are classified in to group A , B and C depending on the fiber diameter .

Group A fibers contain both afferent and efferent myelinated somatic fibers of small, medium and large diameter (1 – 20 μ). They are sub classified into α, β, γ and δ in order of descending diameter and conduction velocity

Group B fibers consist of only small preganglionic myelinated axons of the autonomic nervous system (1 – 3 μ).

Group C fibers consist of small unmyelinated fibers, which are present in visceral afferents, pain and temperature afferents and preganglionic autonomic efferent (2 – 2 . 2 μ).

IMPULSE CONDUCTION :

The action potential originated in the axons is propagated in either direction from its site of origin . The conduction is continuous in unmyelinated and saltatory in mylinated fibers.

MYELINATED FIBERS :

Conduction is much faster in myelinated fibers than in unmyelinated fibers. Myelin thickness is inversely related to inter nodal capacitance and conductance. The conduction velocity increases with increasing myelin in the axon.

As myelin sheath becomes thinner, the inter nodal conductance and capacitance increases in conditions of segmental demyelination such as hypothyroidism or remyelination. This causes greater loss of local current before reaching the next node of Ranvier and fails to activate the nodes of Ranvier . This results in conduction block. The segmental demyelination of smaller fibers may result in continuous conduction instead of saltatory conduction.

UNMYELINATED FIBERS : Impulse conduction in unmyelinated fibers is much slower than in myelinated fibers. The conduction velocity is slow due to the continuous nature of conduction. The conduction velocity further slows down in conditions of focal compression, which may occur due to demyelination or decrease in the diameter of the fibers.

ERLANGER AND GASSER CLASSIFICATION OF MAMMALIAN NERVE FIBERS

Fiber type	Function	Fiber Diameter (µm)	Conduction Velocity (m/s
A			
α	Proprioception; somatic motor	12 - 20	70 – 120
β	Touch, pressure	5 - 12	30 -70
γ	Motor to muscle spindles	3 - 6	15 – 30
δ	Pain, cold, touch	2 - 5	12 – 30
B	Preganglionic autonomic	< 3	3 – 15
C			
Dorsal root	Pain, temperature, some mechanoreception	0.4 – 1.2	0.5 – 2
Sympathetic	Postganglionic sympathetics	0.3 – 1.3	0.7 – 2.3

NUMERICAL CLASSIFICATION (LIOYDS CLASSIFICATION) FOR SENSORY NEURONS

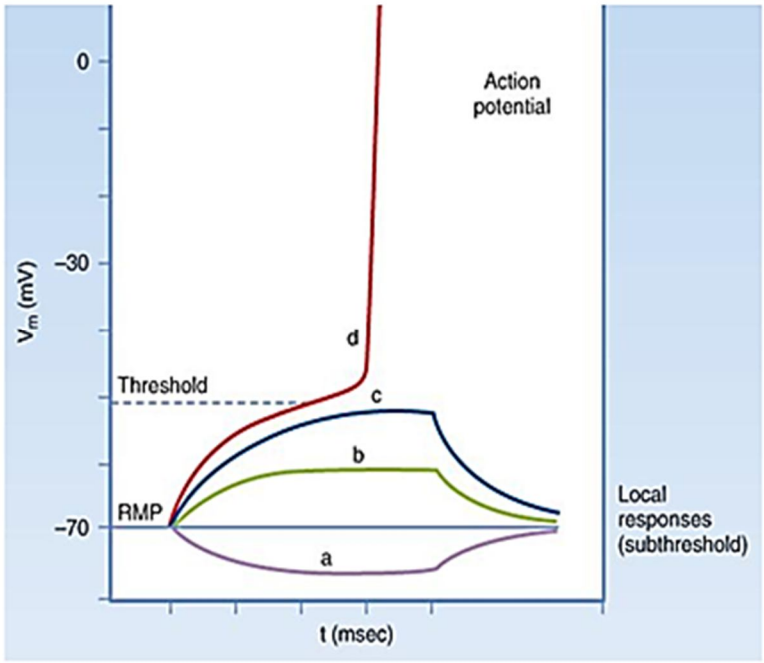
Number	Origin	Fiber type
Ia	Muscle spindle, annulo-spiral ending	Aα
Ib	Golgi tendon organ	Aα
II	Muscle spindle, flower-spray ending; touch, pressure	Aβ
III	Pain and cold receptors; some touch receptors	Aδ
IV	Pain, temperature, and other receptors	Dorsal root C

PHYSIOLOGICAL ASPECTS OF NERVE CONDUCTION

Nerve cells have a low threshold for excitation . The stimulus may be electrical, chemical, or mechanical. The action potentials or nerve impulses are the only electrical responses of neurons and the main language of nervous system (12).

RESTING MEMBRANE POTENTIAL

A nerve at rest is in osmotic equilibrium . The cell membrane contains channels that are selectively permeable to particular ions . The concentration of various ions varies between inside and outside the cell membrane , the concentration of potassium ions is more inside and the concentration of sodium is more outside the cell. The combination of



Responses of an axon to rectangular pulses of hyperpolarizing (a) or depolarizing (b to d) current. Note that when stimulated to threshold (d), the axon fires an action potential. For clarity, only the rising phase of the action potential is shown.

RMP,Resting membrane potential.

above two factors that is the selective permeability of the cell membrane and the different concentration of various ions inside and outside the cell give rise to a potential difference across the membrane (52).

In the resting state, essentially all sodium channels are closed and a small proportion of potassium channels are open . But the voltage gated sodium channels though mostly closed , allow a slight inward leak which is held in check by Na / K ATPase pump. The Chloride ions flow passively and the membrane permeability to Chloride is constant, so it makes no major contribution. So the resting membrane is more permeable to potassium than to sodium and hence the equilibrium potential for potassium largely determines the level of the resting potential (58). The equilibrium potential at which there is no net flow across the membrane is described by Nernst equation as follows

$$E_{ion} = \frac{RT}{nF} \log \frac{[ion]_{outside}}{[ion]_{inside}}$$

R – Gas Constant, F–Faraday Constant, n– Valence of the ion, t-temperature

The eequilibrium potentials vary from cell to cell. The E_k is about -70mV to -90 mV, the E_{Na} is +60 mV, and the E_{cl} is about -70 mV. The resting membrane potential in neurons is about -70mV, which is close to the equilibrium potential for K^+.

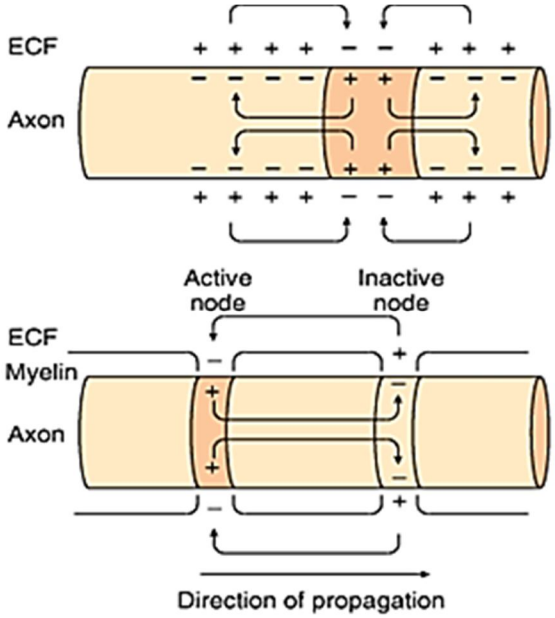
ACTION POTENTIAL

The membrane potential can change in two ways . The decrease in the Membrane potential is such that the interior of the cell becomes more positive with respect to outside is depolarization and the vice versa is hyperpolarization. There are two basic types of changes in membrane potential , propagated and non - propagated . Non – propagated potential is a local potential that occurs due to slight depolarization of the membrane.

During sub threshold depolarization , only a few sodium channels are open and potassium permeability is still greater than sodium. So the depolarization terminates . Once the threshold is exceeded that is decrease in the membrane potential from -70 mV to -55 mV , it results in opening of large number of sodium channels so that sodium permeability exceeds potassium permeability resulting in the generation of propagated action potential .

IMPULSE PROPAGATION ALONG NERVE FIBERS

An action potential can be propagated along the axon . When a segment of axon is depolarized , positive charges from the membrane ahead of and behind the action potential flow into the area of negativity represented by the action potential (" current sink "). By drawing off positive charges , this flow decreases the polarity of the membrane



Local current flow around an impulse in an axon. Top: Unmyelinated axon. Bottom: Myelinated axon. Positive charges from the membrane ahead of and behind the action potential flow into the area of negativity represented by the action potential ("current sink"). In myelinated axons, depolarization jumps from one node of Ranvier to the next (saltatory conduction).

ahead of the action potential. Such electro tonic depolarization initiates a local response, and when the firing level is reached, a propagated response occurs that in turn electrotonically depolarizes the membrane in front of it. In unmyelinated axons, there is continuous conduction which is a slow process.

In myelinated axons, since myelin is an effective insulator, depolarization jumps from one node of Ranvier to the next, with the current sink at the active node serving to electrotonically depolarize the node ahead of the action potential to the firing level. This jumping of depolarization from node to node is called saltatory conduction. It is a rapid process that allows myelinated axons to conduct up to 50 times faster than the fastest unmyelinated fibers (12). The peripheral polyneuropathy is a progressive nerve disorder. It may become chronic disability due to the defect in axons, nerve cell body or myelin sheath (59).

FACTORS THAT AFFECT NERVE CONDUCTION:

1. Physiological factors
2. Technical factors

PHYSIOLOGICAL FACTORS:

A. TEMPERATURE : Nerve temperature is the single most important factor that affects conduction velocity. The nerve conduction velocity is directly related to intraneuronal temperature which in turn depends on internal body temperature.

5 % increase in conduction velocity occurs per degree Celsius rise of body temperature from 30º to 40º range .

Conversely a low temperature , decreases the conduction velocity . For each degree Celsius fall in temperature , the latency increases by 0.3 milli seconds (ms) and velocity decreases by 2.4 meters / seconds (m/s) .

The change in conduction velocity due to alteration in body temperature on sodium channels in the nerves.

AGE : Age significantly affects nerve conduction. The conduction velocity of nerves is low in infants and children. In neonates, it is nearly half of the adult values . It attains the adult value by three to five years of age , then remains relatively stable until 60 years of age, after which it starts declining at a rate of 1.5 % per decade. This is related to gradual loss of larger neurons with ageing.

HEIGHT : an inverse relationship exists between the height of the individual and the velocity of nerve conduction . This is because the shorter nerves conduct faster than the longer nerves of the same age group.

In tall subjects , distal conduction slowing occurs due to greater axonal tapering and lesser myelination.

LIMB: In the upper limb , conduction velocity is higher ; this too is attributed to the length of the nerves . The factors that contribute to the difference in conduction velocity of nerves between the upper and lower limbs are ; abrupt distal axonal tapering in the lower limbs , shorter intermodal distance in the lower limbs. Progressive reduction in axonal diameter in the lower limbs , lower temperature of the feet compared to the hands .

GENDER: Gender is known to affect nerve conduction .

TECHNICAL FACTORS

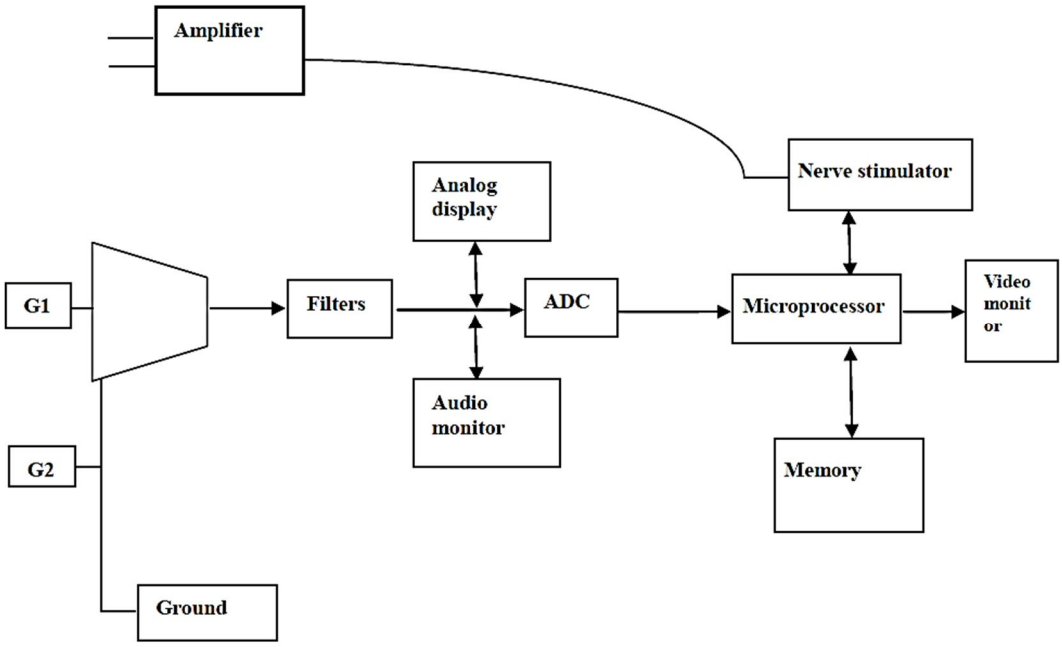
It can be due to a defect in the stimulating system or due to a defect in the recording system.

STIMULATING SYSTEM

Failure of the stimulating system may result in small response or no response.

FAULTY LOCATION OF STIMULATOR

The stimulator may be placed wrongly on the skin surface or the nerve may be stimulated sub maximally . In such cases, the stimulator should be relocated close to the nerve and pressed firmly .



(Schematic diagram showing major components of electro diagnostic equipment. ADC - Analog to digital converter)

FAT OR EDEMA BETWEEN STIMULATOR AND NERVE

In some situations like obesity or edema , the needle electrodes may be used , as the impulse may not reach the target properly.

BRIDGE FORMATION BETWEEN ANODE AND CATHODE

An important source of failure of the stimulating system is the shunting of current between anode and cathode either by sweat or the formation of a bridge by conducting jelly .

RECORDING SYSTEMS

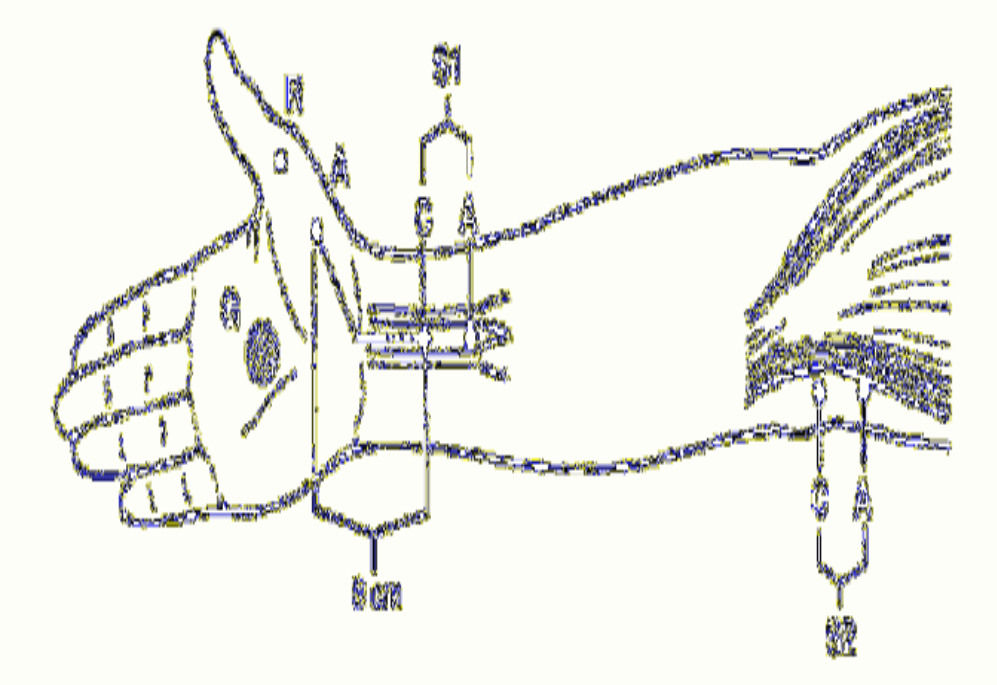
Results may be erroneous if the recording system is defective , especially if the connection is faulty .

DAMAGE IN THE ELECTRODE WIRE

The intactness of the recording system is tested by asking the subject to contract the muscle with the electrode in position . If there is damage in the cable , the stimulus induced muscle twitches causes movement related potentials .

INCORRECT POSITION OF ACTIVE OR REFERENCE ELECTRODE

An Initial positivity preceding the peak of compound muscle action potential suggests incorrect positioning of the active electrode . The recorded potential is also distorted if the reference electrode is located in an active action potential rather than a remote region in relation to muscle action potential .



The picture shows the electrode placement for Median Nerve Motor Conduction study. A-Active electrode, R-Reference electrode, G-Ground electrode, S1-Distal stimulation site, S2-Proximal stimulation site, C-Cathode, A-Anode

WRONGLY CONNECTED PREAMPLIFIER / WRONG SETTING OF GAIN , SLEEP OR FILTER

Amplifier filters can change all the components (amplitude, latency , and duration) of the recorded response .

PRINCIPLES OF NERVE CONDUCTION STUDY

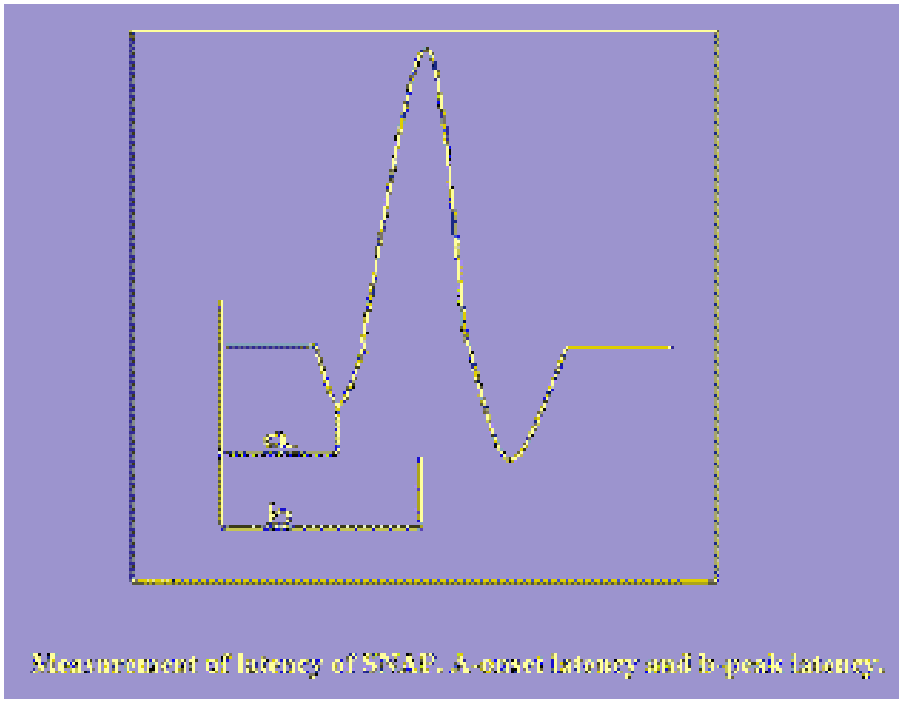
Nerve conduction studies are performed by delivering an electrical stimulus to the nerve which causes depolarization of the nerve and generation of action potential . The cathode of the stimulator delivers the stimulus . The negativity of the cathode draws positive charges away from the axolemma , decreases the trans membrane potential , depolarizes the nerve to threshold and causes generation of an action potential . Surface electrodes are commonly used to record the action potential (58).

PRINCIPLES OF MOTOR NERVE CONDUCTION

In motor nerve conduction study , the motor or mixed nerve is stimulated at two points along its course to record a Compound Muscle Action Potential (CMAP) from a muscle innervated by that nerve .

ELECTRODE PLACEMENT

For recording CMAP , the active recording electrode (A) is placed over the motor point on the belly of the muscle to be studied . The motor point corresponds to motor entry zone of the nerve into the muscle . The reference electrode (R) is placed distal to the active electrode over some



Measurement of latency of SNAP. A-onset latency and b-peak latency.

electrically inactive point such as the muscle tendon . This is called " Belly Tendon montage" .

The ground electrode (G) is placed between stimulating and recording electrodes (52).

STIMULATION

The nerve is stimulated by a supramaximal stimulus keeping the cathode close to the active electrode . A supramaximal stimulus will reliably stimulate all fibers in a nerve including the fastest fibers in the nerve . Surface stimulation of the healthy nerve requires a square wave pulse of 0.1 ms duration with an intensity of 5-40 mA (58) . Supramaximal stimulation is given by progressively increasing the stimulus intensity until there is no increase in the amplitude of CMAP , then increasing the intensity further by 20 – 50 % for the final stimulation (58).

COMPOUND MUSCLE ACTION POTENTIAL (CMAP)

The response following stimulation of the nerve is a biphasic action potential with initial negative deflection provided the active electrode is placed properly over the motor point of the muscle . When the electrode is away from the motor point the initial deflection will be positive which is followed by main negative deflection . CMAP is the summated , compounded response of all the muscle fibers that lie within the recording area of the electrode and have responded to the electrical stimulation of the nerve (51)(58).

PARAMETERS MEASURED IN MOTOR NERVE CONDUCTION STUDY

1. Distal motor latency
2. Proximal motor latency
3. Amplitude
4. Duration
5. Motor nerve conduction velocity

1. DISTAL MOTOR LATENCY

The time taken for an impulse to travel from the distal site of nerve stimulation is the distal motor latency. It is the time in milliseconds from the stimulus artifact to the first negative deflection of CMAP . It is the summation of several events such as the time taken to depolarize the nerve called " utilization time " , the time taken for the impulse to travel from the site of stimulation to the motor end plate and the " residual latency " which includes neuromuscular transmission time plus the propagation time along the muscle membrane.

2. PROXIMAL MOTOR LATENCY

It is the time taken for an impulse to travel from the proximal site of nerve stimulation. Onset latency is a measure of conduction in the fastest conducting motor fibers.

3. AMPLITUDE OF CMAP :

The amplitude is measured from the baseline to the height of the negative peak (base – to – positive peak (peak) or from the height of the negative peak to the depth of the peak – to – peak) . The amplitude corresponds to the number of nerve fibers .

4. DURATION

The duration of CMAP is measured from the onset to the negative peak or positive peak or the final return of wave form to the baseline . It correlates with the density of small fibers .

The area under the CMAP can also be measured by computer analysis . The area under the curve is useful in distinguishing between true conduction block and loss of amplitude due to temporal dispersion .

TEMPORAL DISPERSION

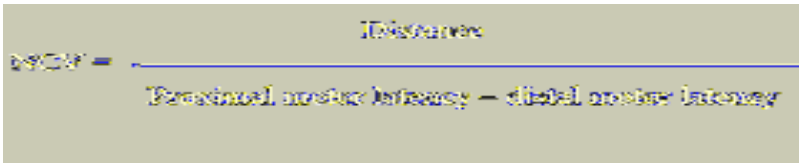
The peripheral nerve contains a number of axons of different sizes and different conduction velocities . Due to this difference , when a nerve is stimulated at more proximal sites from recording electrode there is dispersion of CMAP as the volley of impulses arriving at the recording electrode becomes less synchronized (58) . This is the phenomenon of temporal dispersion which causes a decrease in amplitude and increase in duration of CMAP on proximal stimulation .

Usually there is 20 % decrease in negative peak amplitude on proximal stimulation and decrease of > 20 % suggests either sub maximal proximal stimulation or spread of the stimulus to adjacent nerves or a demyelinating lesion in the nerve between the two sites (51). But there is less change in area under the curve since the same number of nerve fibers is eventually stimulated .

5. MOTOR NERVE CONDUCTION VELOCITY

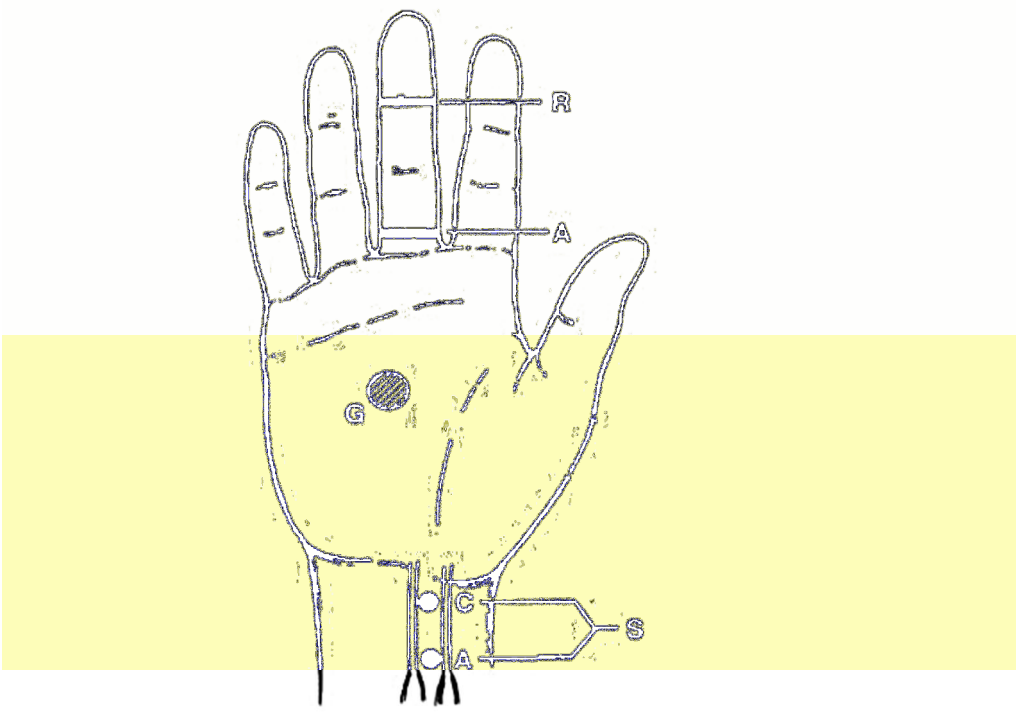
Nerve conduction velocity (NCV) can be determined by stimulating the nerve at two sites separated by a known distance . NCV is calculated by measuring the distance in millimeter between two points of stimulation , which is divided by the latency difference in millisecond.

It is expressed in m / s.



PRINCIPLES OF SENSORY NERVE CONDUCTION

Sensory nerve conduction studies are performed by stimulating the nerve and recording the sensory nerve action potential (SNAP) directly by electrodes placed over the nerve . It can be done either antidromically or orthodromically .



Electrode Placement

Electrode placement for Antidromic Sensory Nerve Conduction of Median nerve. A-Active electrode, R-Reference electrode, G-Ground electrode, S-stimulator, C-Cathode, A-Anode.

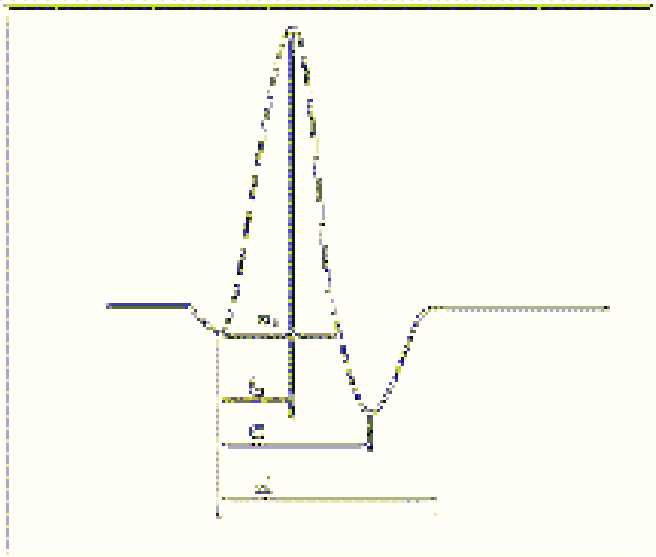
In orthodromic conduction , the distal portion of the nerve is stimulated and sensory nerve action potential is recorded proximally along the nerve . It is orthodromic because the impulses are travelling in the same direction as would sensory impulses .

In antidromic conduction , the stimulation is given at proximal part of the nerve and sensory nerve action potential is recorded at the distal part of the nerve that is the impulses travel in the opposite direction . Antidromic techniques are preferred over orthodromic techniques .

ELECTRODE PLACEMENT

Ring electrodes are used for orthodromic digital nerve stimulation or recording antidromic responses . The active electrode is placed proximally and reference electrode is placed distal to the active electrode . The ground electrode is placed between active and stimulating electrode . The distance between the active electrode and reference electrode is critical because the summated SNAP amplitude is the algebraic total of the electrical activity recorded at the two electrodes . If the two electrodes are close , both become active which distorts the waveform and decreases the amplitude of action potential. (60)

The inter electrode distance should be at least 3 cm for the potential to clear the active electrode before the activity begins at the reference electrode .



Μετασωματικό σε ζωνώδη μορφή, σχιστό με διάπλαση διάτμησης. Τα ανακρυσταλλωμένα φύλλα από λεπτόκοκκο χλωρίτη, ασβεστίτη μετατρέπονται σε γραφίτη χαλαζιακά ενδιάμεσα με λεπτόκοκκο ασβεστίτη.

Sensory nerve conduction can be done by both stimulating and recording from a pure sensory nerve, stimulating a mixed nerve and recording from its sensory branch (e.g. Median antidromic digital) , or stimulating a sensory nerve and recording from a mixed nerve (e.g. Median orthodromic digital).

SENSORY NERVE ACTION POTENTIAL

SNAP is a triphasic potential with an initial and a terminal positivity and a negative deflection in the middle . The negative deflection indicates the time of arrival of the impulses beneath the active electrode .

Parameters measured in sensory nerve conduction study :

1. Latency
2. Amplitude
3. Duration
4. Nerve conduction velocity :

1**. LATENCY**

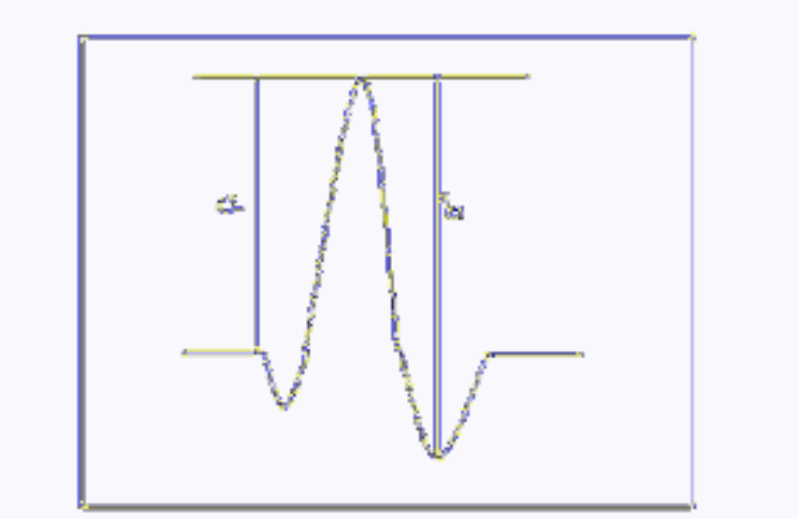
The latency of orthodromic potential is measured from the stimulus artifact to the initial positive or subsequent negative peak .(61) The initial positivity is frequently absent in antidromic potential.

2. **AMPLITUDE**

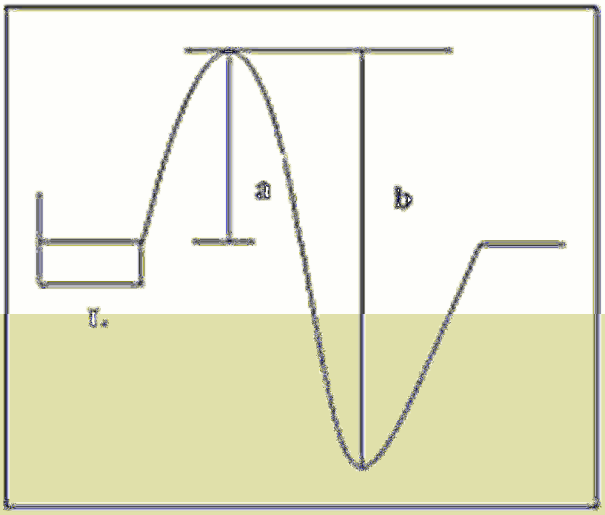
The amplitude is measured from baseline to negative peak or from positive to negative peak . The amplitude of antidromic potential is larger than orthodromic potential since in antidromic stimulation the recording electrodes are closer to the nerve especially in digital nerves (62)

3. **DURATION**

Duration is measured from the positive peak to the intersection between the descending phase and the baseline or to the negative peak or subsequent positive peak or return to the baseline .



Measurement of SNAP amplitude. a-base-to-peak amplitude and b-peak-to-peak amplitude.



Measurement of CMAP latency and amplitude. L-onset latency, a-base-to-peak amplitude and b-peak-to-peak amplitude.

4. SENSORY NERVE CONDUCTION VELOCITY (SNCV) :

SNCV can be determined by stimulation at a single site unlike motor nerve conduction velocity . Because the residual latency as mentioned earlier in motor nerve conduction is not applicable to sensory nerve conduction. SNCV is calculated by dividing the distance in millimeter between the center of stimulating electrode and recording electrode by the latency in milliseconds .

$$\text{SNCV (m/s)} = \frac{\text{Distance}}{\text{Latency}}$$

CLINICAL APPLICATIONS OF NERVE CONDUCTION STUDY

Nerve conduction study is not only useful in identifying the nerve damage but also it gives an idea about the pattern of nerve damage . Two major types of pathological changes affect nerve conduction , Axonal degeneration and Demyelination.

AXONAL DEGENERATION

Axonal loss can occur as a consequence of neuronal cell death or injury to distal segment resulting in discontinuation from the neuronal cell body or in the most distal part of the axon as a ‘ dying back phenomenon ’(63). The pathological changes are disappearance of cytoskeletal elements , discontinuation of axolemma and secondary disintegration of myelin sheath but not the Schwann cell (58).

ELECTROPHYSIOLOGICAL CHANGES

Pure axonal loss produces reduction in the amplitude of compound muscle action potential and sensory nerve action potential with preservation of conduction velocity , because the surviving axons conduct at their normal velocity (52).

DEMYELINATION

Here the myelin sheath or the Schwann cell is primarily affected and the axon is intact . It can occur in the Para nodal region or can affect the whole inter nodal segment called ' Segmental demyelination ' .

MEDIAN NERVE - MOTOR COMPONENT

The elbow - wrist segment of median nerve was tested . The basic principle involves the stimulation of median nerve by a supramaximal stimulus at two points that is at wrist and elbow to record a Compound Muscle Action Potential (CMAP) of Abductor Pollicis Brevis muscle supplied by the nerve using surface electrodes.

ELECTRODE PLACEMENT: 3 electrodes were used

1. Active or Recording electrode was placed close to the motor point of Abductor Pollicis Brevis muscle (halfway between the midpoint of the distal wrist crease and the first meta carpo phalangeal joint).

2. Reference electrode was placed 3 cm distal to active electrode at first metacarpophalangeal joint over the tendon of Abductor Pollicis Brevis muscle .

3.Ground electrode was placed between stimulating and active electrode .

A supramaximal stimulus was used to stimulate the nerves . Surface stimulation was performed as per the following steps :

Site 1: At wrist , between the tendons of palmaris longus and flexorcarpi radialis approximately 1 cm proximal to the most distal wrist crease.

Site 2: At elbow crease , medial to biceps tendon and brachial artery

MEDIAN NERVE – SENSORY COMPONENT

Median nerve is stimulated at wrist and sensory nerve action potentials are recorded from digital nerve of index finger antidromically using ring electrodes.

ELECTRODE PLACEMENT

1. Active or Recording electrode was placed at proximal inter phalangeal joint of index finger.

2. Reference electrode was placed at distal inter phalangeal joint of index finger.

The inter electrode distance should be at least 3 cm.

3. Ground electrode was placed between stimulating and active electrode.

NERVE STIMULATION

A supramaximal stimulus was used to stimulate the nerves.

Site : At wrist , between the tendons of palmaris longus and flexor carpi radialis approximately 1 cm proximal to the most distal wrist crease .

PARAMETERS MEASURED IN NERVE CONDUCTION STUDY

1. Proximal and distal latency of the action potential
2. Amplitude of the action potential
3. Nerve Conduction Velocity

OBSERVATION

Express the nerve conduction velocity in meters per second (m / s).

PRECAUTIONS TAKEN

1. The subject was properly instructed and motivated to provide full cooperation .
2. The subject was made fully relaxed .
3. The room was quiet and comfortable .
4. The subject was grounded properly .

Recording of nerve conduction study was done to assess the velocity of conduction of impulses in the nerve . The important nerves tested were median , ulnar nerves in both the upper limbs .

MEDIAN NERVE

The median nerve (C5 - T1) is a mixed nerve , that is it contains both motor and sensory components . It supplies the flexors of the forearm and thenar muscles of hand . It is sensory to the lateral aspect of the palm and the dorsal surface of terminal phalanges . It has no innervation in the upper arm . In the forearm it supplies pronator teres , flexor carpi radialis , palmaris longus , flexor digitorum superficialis , flexor digitorum profundus , flexor pollicis longus and pronator quadratus . The nerve then passes through the carpal tunnel to enter the hand , where it supplies lumbricals I and II, opponens pollicis , flexor pollicis brevis and abductor pollicis brevis.

ENTRAPMENT NEUROPATHY OF MEDIAN NERVE

The entrapment (compression) neuropathy of the median nerve occurs commonly during its course in the carpal tunnel. In fact, the carpal tunnel syndrome is the commonest entrapment neuropathy seen in the neurology clinic. Pronator teres syndrome of the median nerve (entrapment of the nerve between the heads of the pronator teres , through which the nerve descends into the forearm from the arm) also occurs occasionally. Hypothyroidism is commonly included as an important risk factor for carpal tunnel syndrome (CTS) yet this association between the two is not defined in any study (64). Some reports suggest that axonal demyelination

can result in peripheral mononeuropathy, polyneuropathy or entrapment neuropathies in hypothyroidism (65).

In carpal tunnel syndrome , the conduction of the median nerve decreases . The nerve conduction decreases distal to the site of compression. The motor distal , F –minimal and sensory latencies , amplitudes and conduction velocities between the median and ulnar nerves were compared to establish the diagnosis of median nerve compression at wrist (conventional conduction studies) . The sensory and motor nerve conduction studies (NCS) of the median nerve segment across the wrist compared to another nerve such as ulnar nerve , are the most sensitive and accurate techniques for making a diagnosis of Carpal tunnel syndrome (66).

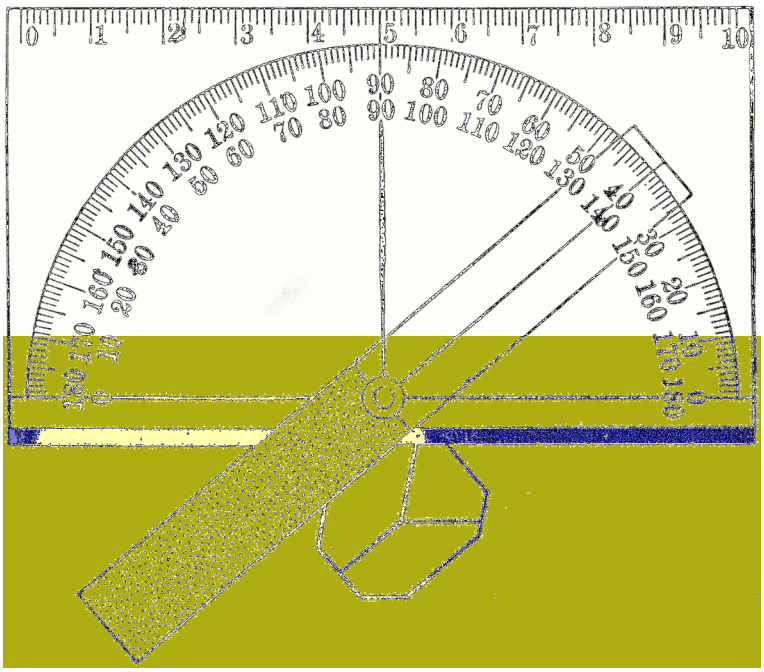
CAUSES OF CARPAL TUNNEL SYNDROME

The common causes are : Rheumatoid arthritis, overuse of wrist, hypothyroidism, acromegaly.

ULNAR NERVE

The ulnar nerve arises from C7 - T1 through the medial cord of brachial plexus . It does not supply any muscle in the upper arm . It passes through the condylar groove in the elbow , to enter the forearm where it passes through the cubital tunnel. Here it supplies the flexor carpi ulnaris . Then it supplies the flexor digitorum profundus III and IV . At the wrist , it passes through Guyton ’s canal where it bifurcates to form a superficial

GONIOMETER


0 1 2 3 4 5 6 7 8 9 10
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180

sensory and a deep motor branch . The motor branch supplies hypo thenar muscles and abductor pollicis , medial half of flexor pollicis , interosseous and third and fourth lumbricals .

ULNAR NEUROPATHY

Ulnar nerve neuropathy can occur at the elbow , the distal forearm , and wrist (50).

GONIOMETRY

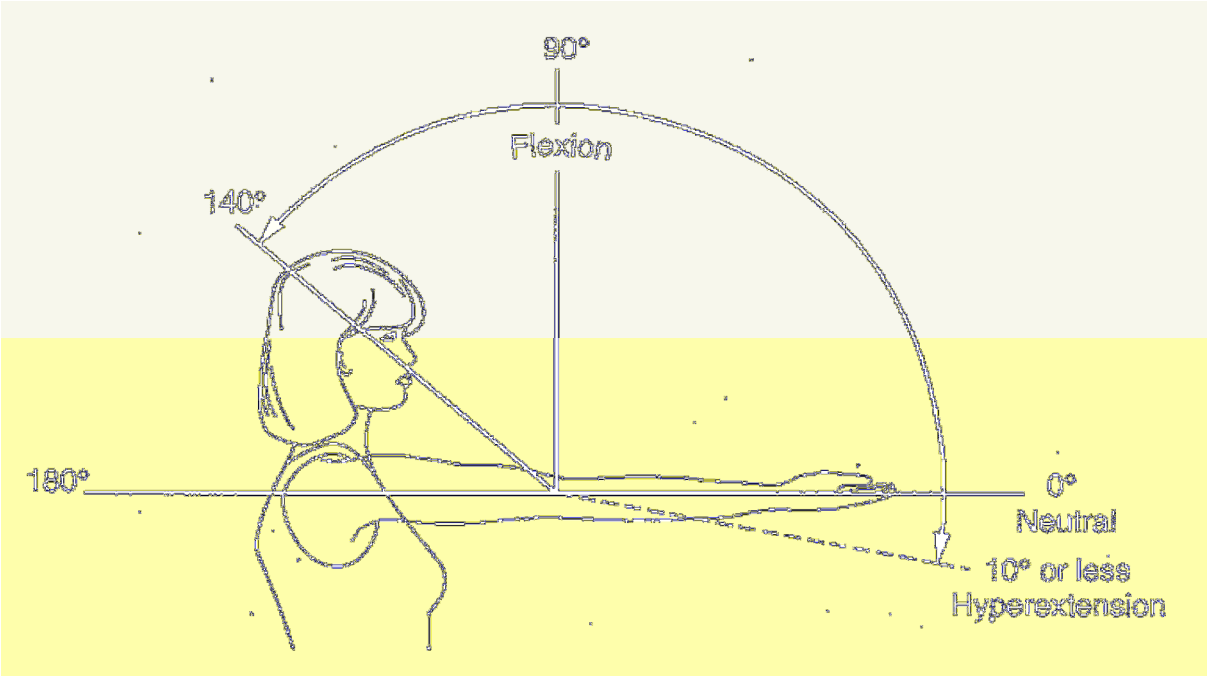
Historically , goniometry developed over the last 60 years in conjunction with the rapid growth of the field of physical medicine and rehabilitation . A recent article by Smith provides an interesting account of this development (67). Also the recent publication by Norkin and Whites gives current and complete descriptions (68).

Goniometer is a device used to measure joint angles or range of movement. In musculoskeletal disorders, goniometer is used to measure the range of movement (in degrees) of joints for either active or passive joint range. Using a goniometer we can quantify posture, including measuring joint angles during performance of a task.

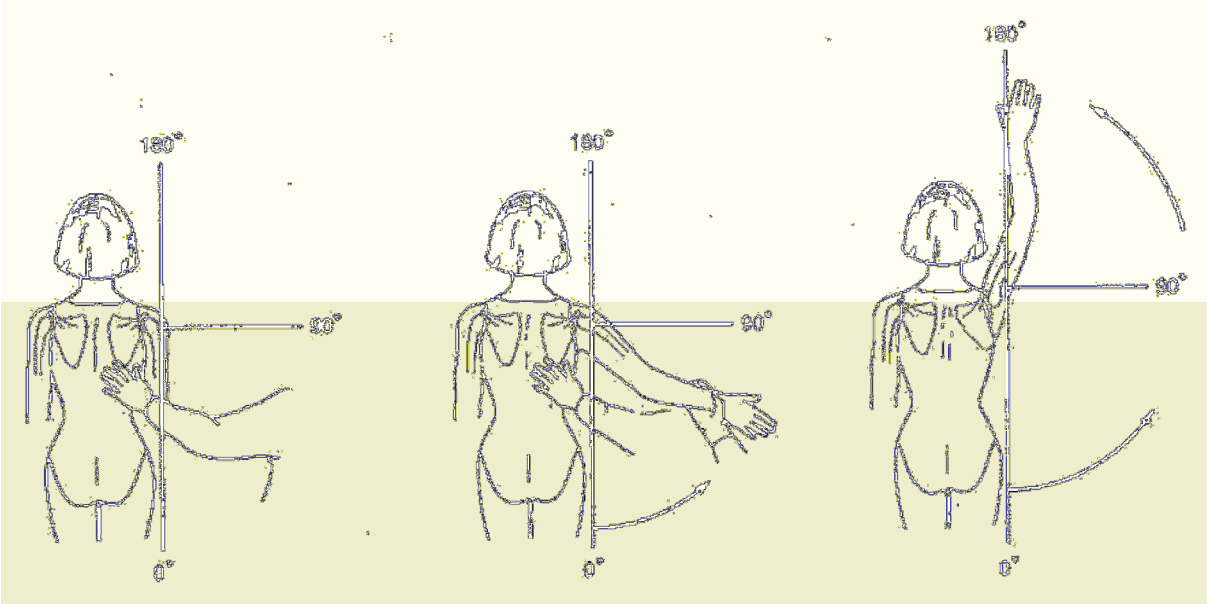
Knowing the joint angle associated with a task can help ergonomist to make more specific design recommendations or to compare worker posture before and after changes have been made.

A goniometer can also measure progress in return of range of movement during recovery. A traditional goniometer is a protractor with

ELBOW JOINT ROM



SHOULDER GIRDLE MOVEMENTS & ROM



extending arms. To use a goniometer, first we have to align the fulcrum of the device with the fulcrum or the joint to be measured . Then secondly align the stationary arm of the device with the limb being measured. Thirdly hold the arms of the goniometer in place while the joint is moved through its range of motion. The degree between the endpoints represents the entire range of movements.

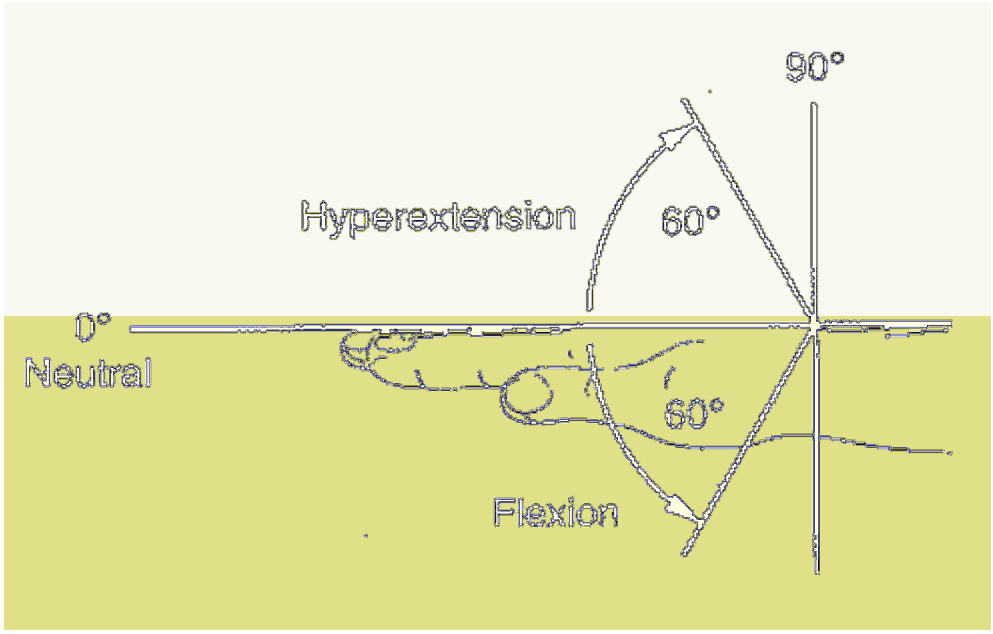
PROCEDURE

The stationary portion of the body was stabilized . This was the part of the body that was proximal (closer to the midline of the body) to the joint that was tested. It was important that the patient did not move his body while moving the joint, this step isolated the joint movement for a more accurate measurement. Goniometer reading was noted before removing it from the patient 's body . Accurate reading of the degree of motion on the goniometer was ensured and that I consistently used the same stationary and movable landmarks on the body when measuring , to ensure the consistency . It was ensured to record the range of motion of the joint .

GONIOMETER MEASUREMENTS

With the goniometer measuring the angle through which the participant can move at the particular joint . The goniometer has a central area or disc where the angles of movement (range of motion) are read . It has two arms : a stationary arm and a moving arm . Basically , the goniometer is centered on the axis of rotation . The stationary arm was

WRIST JOINT ROM



placed in the starting position and did not move as the participant moved through the range of motion . The moving arm starts in the same position as the stationary arm , but as the participant moves through the Range Of Movement it follows until no more movement is possible .

The angle of movement from the stationary arm to the movement arm is read off the central disc and reported as the Range Of Movements . The position the body was supposed to be in for movement and any stabilization issues . The participant was asked to warm – up before performing the flexibility tests (10) .

RELIABILITY

Reliability in goniometry simply means the consistency or the repeatability of the ROM measurements , that is , whether the application of the instrument and the procedures produce the same measurements consistently under the same conditions. (69). Gajdosik and Lusin (70) and Gajdosik et al (71) have demonstrated that even complex movements can be measured reliably when the measurement procedures are controlled strictly. Lawrence confirmed the validity of goniometry at the knee by comparing goniometric measurements with measurements from radiographs of cadavers and patients (72) .

Joint/Segment	Movement				
Elbow	Flexion	140	145	145	145
	Hyperextension	0	0	0	0-10
Forearm	Pronation	80	90	90	80
	Supination	80	85	90	90
Wrist	Extension (Dorsiflexion)	60	70	70	50
	Flexion (Palmar flexion)	60	90	-	60
	Radial Deviation	20	20	20	20
	Ulnar Deviation	30	30	35	30
Shoulder	Flexion	180	170	130	180
	Hyperextension	50	30	80	60
	Abduction	180	170	180	180
	Adduction	50	-	-	-
Shoulder w/ Abducted Arm	Internal Rotation	90	90	70	60-90
	External Rotation	90	90	70	90
	Horizontal Adduction	-	-	-	135
	Horizontal Adduction	-	-	-	45
Hip	Flexion	100	120	125	120
	Hyperextension	30	10	10	30
	Abduction	40	45	45	45
	Adduction	20	-	10	0-25
Extended Hip	Internal Rotation	40	35	45	40-45
	External Rotation	50	45	45	45
Knee	Flexion	150	120	140	130
Ankle	Plantar flexion	20	45	45	50
	Dorsiflexion	30	15	20	20
Cervical Spine	Flexion	60	-	-	40
	Hyperextension	75	-	-	40
	Lateral Flexion	45	-	-	45
	Rotation	80	-	-	50
Lumbar-thoracic Spine	Flexion	45-50	-	-	45
	Hyperextension	25	-	-	20-35
	Lateral Flexion	25	-	-	30
	Rotation	30	-	-	45

MATERIALS & METHODS

MATERIALS AND METHODS

INCLUSION CRITERIA

Newly diagnosed hypothyroid women .

Hypothyroid women who were newly diagnosed and not started on treatment were included in this study .

EXCLUSION CRITERIA

- Hypothyroid patients on treatment (already diagnosed)
- Alcoholism
- Diabetes Mellitus
- Neuromuscular disorders
- Leprosy
- Drug induced Neuropathy
- Malignancy
- HIV
- Liver disease
- Kidney disease
- Myopathy.

STUDY PERIOD From July 2015 to March 2016 .

SAMPLE SIZE A total of 100 women were included in this study. Out of 100 women 50 were newly diagnosed hypothyroid women and 50 were normal euthyroid women.

PLACE OF STUDY Coimbatore Medical College Hospital, Coimbatore.

PATIENT SOURCE FOR STUDY : 50 numbers of newly diagnosed hypothyroid women, more than 20 years of age were selected from the Female Medical Out Patient Department (O P D) and 50 numbers of euthyroid age matched women were selected as controls from Female Medical Out Patient Department, of Coimbatore Medical College Hospital. Coimbatore.

METHODOLOGY

Patients and controls were picked up from Female Medical O P D, and height and weight measurements were done at Non Communicable Diseases Out Patient Department, Thyroid Function Tests were done at Clinical Biochemistry Laboratory, Coimbatore Medical College Hospital, Coimbatore, using Microwell ELISA technique with ERBA Thyrokit from ERBA Diagnostics. A complete NCS was done in all the subjects with RMS – EMG - EP Mark II using standard protocols and settings. 1- cm

disc surface electrodes were used with surface stimulators . Three types of electrodes were used i.e. active , reference and ground . The ground electrode served as a zero voltage reference point (73) . The nerve conduction velocities to the electrical stimulation of the nerves of normal euthyroid women were compared with hypothyroid women . Goniometry instrument was used to find out the degree and range of movement of joints , at the Neurology Laboratory , Department of Neurology in Coimbatore Medical College Hospital , Coimbatore.

RESULTS

RESULTS

All the subjects (both euthyroid and hypothyroid) included in the present study were well nourished with Normal dietary habits . On clinical examination , they had no evidence of nutritional deficiencies .

CHARACTERISTICS OF CONTROL AND STUDY POPULATIONS

The characteristics of control and study populations are presented in Table 1 . Among the individuals included in the present study , the mean age of the study group was found to be 36.10 ± 8. 048 years with the range of 20 to 60 years . All the individuals included in the study were women . The mean height of the study group was found to be 1. 5531 ± 0 .05678 meters . The mean weight was found to be 65.4080 ± 7.80274 kilograms . The mean Body Mass Index (BMI) was found to be 26.97 ± 2.925.

TABLE:1 Data showing (mean ± SD) values of different parameters among study population

Statistics					
		Age yrs	Ht	Wt	BMI
N	Valid	100	100	100	100
	Missing	0	0	0	0
Mean		36.10	1.5531	65.4080	26.97
Std. Deviation		8.048	.05678	7.80274	2.925

Table : 2. Comparing the mean ± SD of different parameters in Hypothyroid women and Euthyroid women . (Note : 1 = Hypothyroid women . 2 = Euthyroid women)

	1 OR 2	N	Mean	Std. Deviation	Std. Error Mean
Age yrs	1	50	34.66	6.355	.899
	2	50	37.54	9.287	1.313
Ht	1	50	1.5468	.05751	.00813
	2	50	1.5594	.05589	.00790
Wt	1	50	68.7500	6.55141	.92651
	2	50	62.0660	7.56923	1.07045
BMI	1	50	28.60	2.449	.346
	2	50	25.34	2.421	.342

Abbreviations : yrs = years . Ht = Height . Wt = Weight . BMI = Body Mass Index .

The above table displays the mean age ± SD of hypothyroid women marked as 1 was found to be 34.66 ± 6.355 years and the mean age ± SD of normal euthyroid women marked as 2 was found to be 37.54 ± 9.287 . The mean height ± of hypothyroiod women was found to be 1.5468 ± 0.05751 meters and the mean height ± of euthyroid women was found to be 1.5594 ± 0.05589 .

The mean weight of hypothyroid women was found to be 68.7500 ± 0.92651 kilograms and that of euthyroid women was found to be

62.0660 ± 7.56023 . The Body Mass Index of hypothyroid women was found to be 28.60 ± 2.449 . BMI of euthyroid women was found to be 25.34 ± 2.421 .

THE NERVE CONDUCTION PARAMETERS:

TABLE : 3, Showing the Nerve conduction Parameters (Note : 1 = Hypothyroid women . 2 = Euthyroid women)

NCS	1 OR 2	N	Mean	Std. Deviation	Std. Error Mean
RT MN MCV	1	50	48.7412	3.64279	.51517
	2	49	54.3422	3.64267	.52038
RT MN SCV	1	50	48.443600	3.7253630	.5268459
	2	50	52.991000	8.5447036	1.2084036
F WAVE	1	50	30.045200	1.9217671	.2717789
	2	50	32.452800	2.4383493	.3448347
RT UN MCV	1	50	49.226800	4.8491802	.6857776
	2	50	56.635600	5.3236393	.7528763
RT UN SCV	1	50	48.470000	5.0615493	.7158112
	2	50	53.713200	5.4610791	.7723132
F WAVE	1	50	30.167800	2.9244635	.4135816
	2	50	34.757840	2.7299220	.3860693
LT MN MCV	1	50	48.880400	5.1410981	.7270611
	2	50	55.215200	4.9292187	.6970968
LTMNSCV	1	50	47.342600	8.7771526	1.2412768
	2	50	52.139800	5.2051640	.7361214
F WAVE	1	50	30.326600	2.0352522	.2878281
	2	50	32.425800	2.1986759	.3109397
LT UN MCV	1	50	48.897200	5.5795849	.7890725
	2	50	54.666600	4.6017927	.6507918
LT UN SCV	1	50	49.2036	4.23964	.59958
	2	50	53.8632	3.75992	.53173
F WAVE	1	50	29.999200	1.9428097	.2747548
	2	50	32.861200	2.4731700	.3497591

Abbreviations : Rt = Right . Lt = Left . MN = Median Nerve . UN = Ulnar Nerve . MC = Motor Conduction Velocity . SC= Sensory Conduction Velocity .

The bilateral median nerve and the ulnar nerves were included in this study . The motor component , sensory component and F wave were recorded in this study . The nerve conduction velocity was compared between the hypothyroid and euthyroid women . F wave evaluates the conduction velocity of nerves between the limb and spinal cord (74) .

The right median nerve mean ± motor conduction velocity in hypothyroid women was found to be 48.741 ± 3.642 meters / second and euthyroid women had mean ± right median (nerve) motor nerve conduction velocity of 54.342 ± 3.643 m / s . The mean ± value on the right side median nerve sensory component in hypothyroid women was found to be 48.443 ± 3.725 meters / second . On the contrary the mean ± right median nerve sensory component conduction velocity found in normal euthyroid women was 52.991 ± 8.544 m / s . The F wave response value for right median nerve was found to be 30.045 ± 1.921 m/s among the hypothyroid women and the same F wave response in euthyroid women was found to be 32.452 ± 2.438 m / s .

The right ulnar nerve motor conduction velocity in hypothyroid women was found to be 49.226 ± 4.849 m / s and same was found to be 56.635 ± 5.323 m / s in the euthyroid women . The sensory nerve conduction velocity of the ulnar nerve on the right side in hypothyroid women was found to be 48.470 ± 5.061 m / s, which among the

euthyroid women was found to be 53.713 ± 5.461 m / s . The F wave response of right ulnar nerve of the hypothyroid women was found to be 30.167 ± 2.924 m / s and the same was found to be 34.757 ± 2.729 m / s in the euthyroid women .

The left median (nerve) motor nerve conduction velocity among hypothyroid women was found to be 48.880 ± 5.141 m / s . same was found to be 55.215 ± 4.929 m / s among the euthyroid women . The left median nerve sensory component velocity was 47.342 ± 8.777 m / s in the hypothyroid women , which was found to be 52.139 ± 5.205 m / s among the euthyroid women . The F wave response of left median nerve among the hypothyroid women was 30.326 ± 2.035 m / s and the same was found to be 32.425 ± 2.198 m / s in the euthyroid women .

The left ulnar nerve motor velocity among the hypothyroid women was found to be 48.897 ± 5.579 m / s , whereas the same was found to be 54.666 ± 4.601 m / s in the euthyroid women . The left ulnar nerve sensory conduction was found in hypothyroid women to be 49.203 ± 4.236 m / s and which was found to be 53.863 ± 3.759 among the euthyroid women. The F wave response for left ulnar nerve among the hypothyroid women was 29.999 ± 1.942 m / s and it was found to be 32.861 ± 2.473 m / s among the euthyroid women . Motor polyneuropathy was noticed in patients with hypothyroidism (75) .

GONIOMETRIC PARAMETERS :

TABLE 4. GONIOMETRIC MEASUREMENTS

RT SHOULD	1	50	62.90	10.080	1.425
	2	50	63.30	10.126	1.432
LT.SHOULD	1	50	63.22	9.577	1.354
	2	50	63.64	10.251	1.450
RT.ELBOW	1	50	67.36	12.292	1.738
	2	50	68.40	9.129	1.291
LT.ELBOW	1	50	68.02	12.038	1.702
	2	50	67.98	9.542	1.349
RT WRIST	1	50	61.72	12.177	1.722
	2	50	60.38	14.781	2.090
LT WRIST	1	50	61.60	12.360	1.748
	2	50	60.82	14.420	2.039
RT MCP	1	50	63.40	11.270	1.594
	2	50	64.62	10.713	1.515
LT MCP	1	50	63.80	11.375	1.609
	2	50	64.68	9.942	1.406

Table 4 shows the goniometric measurements - comparison between hypothyroid and euthyroid women .

The various goniometric measurements include the joints of upper limb , namely right shoulder , left shoulder , right elbow , left elbow , right wrist, left wrist, right metacarpophalangeal joint and left metacarpophalangeal joint. Goniometry measures the degree and range of movements of joints. In this study the degree of movement of above joints was measured . There was not of much significant difference in the joint movements between the hypothyroid and euthyroid women .

The mean joint movement of right shoulder among the hypothyroid women was found to be 62.90 ± 10.080 degrees when compared to that of the euthyroid women , whose value was found to be 63.30 ± 10.126 . The left shoulder joint movement was found to be 63.22 ± 9.577 degrees in hypothyroid women and the same was found to be 63.64 ± 10.251 degrees among the euthyroid women . The right elbow joint movement was found to be 67.36 ± 12.292 degrees in the hypothyroid women and the same was found to be 68.40 ± 9.129 degrees in the euthyroid women.

The left elbow joint movement of hypothyroid women was found to be 68.02 ± 12.038 degree , and the same was found in euthyroid women to be 67.98 ± 9.542 degree . The right wrist joint movement was found to be 61.72 ± 12.177 degree in hypothyroid women , and it was found to be 60.38 ± 14.781 degree in euthyroid women . The left wrist joint movement was found to be 61.60 ± 12.177 in hypothyroid women and it was found to be 60.82 ± 14.420 degree in euthyroid women .

As of right metacarpophalangeal joint movement in hypothyroid women is concerned , it was found to be 63.40 ± 11.270 degree and on the euthyroid women it was found to be 64.62 ± 10.713 degree . The left metacarpophalangeal joint movement was found to be 63.80 ± 11.375 degree among the hypothyroid women and the same was found to be 64.68 ± 9.942 degree .

Statistical analysis was done using SPSS software version 16 . The independent ‘t’ test analysis showed that there was significant difference in weight and BMI between hypothyroid and euthyroid women .

Table : 5. The table showing the statistically significant parameters.

	Independent Samples Test								
	Levene's Test for Equality of Variances		t-test for Equality of Means						
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
								Lower	Upper
Age yrs	8.915	.004	-1.810	98	.073	-2.880	1.592	-6.038	.278
			-1.810	86.639	.074	-2.880	1.592	-6.043	.283
Ht	.107	.745	-1.111	98	.269	-.01260	.01134	-.03511	.00991
			-1.111	97.920	.269	-.01260	.01134	-.03511	.00991
Wt	.181	.671	4.721	98	.000	6.68400	1.41573	3.87453	9.49347
			4.721	96.025	.000	6.68400	1.41573	3.87381	9.49419
BMI	.539	.465	6.693	98	.000	3.260	.487	2.293	4.227
			6.693	97.987	.000	3.260	.487	2.293	4.227
RT SHOULD	.023	.881	-.198	98	.843	-.400	2.021	-4.410	3.610
			-.198	97.998	.843	-.400	2.021	-4.410	3.610
LT.SHOULD	.067	.796	-.212	98	.833	-.420	1.984	-4.357	3.517
			-.212	97.550	.833	-.420	1.984	-4.357	3.517
RT.ELBOW	.018	.893	-.480	98	.632	-1.040	2.165	-5.337	3.257
			-.480	90.447	.632	-1.040	2.165	-5.342	3.262
LT.ELBOW	.003	.960	.018	98	.985	.040	2.172	-4.271	4.351
			.018	93.144	.985	.040	2.172	-4.274	4.354
RT WRIST	1.704	.195	.495	98	.622	1.340	2.708	-4.035	6.715
			.495	94.537	.622	1.340	2.708	-4.037	6.717
LT WRIST	.686	.410	.290	98	.772	.780	2.686	-4.550	6.110
			.290	95.760	.772	.780	2.686	-4.552	6.112
RT MCP	.178	.674	-.555	98	.580	-1.220	2.199	-5.584	3.144
			-.555	97.749	.580	-1.220	2.199	-5.584	3.144

LT MCP	.375	.542	-.412	98	.681	-.880	2.136	-5.120	3.360
			-.412	96.274	.681	-.880	2.136	-5.121	3.361
RTMNMC	1.724	.192	-7.649	97	.000	-5.60104	.73225	-7.05437	-4.14772
			-7.649	96.960	.000	-5.60104	.73225	-7.05437	-4.14772
RTMN SC	1.865	.175	-3.450	98	.001	-4.5474000	1.3182586	-7.1634413	-1.9313587
			-3.450	66.979	.001	-4.5474000	1.3182586	-7.1786707	-1.9161293
F WAVE	.608	.438	-5.484	98	.000	-2.4076000	.4390612	-3.2789026	-1.5362974
			-5.484	92.926	.000	-2.4076000	.4390612	-3.2794977	-1.5357023
RTUNMC	.133	.716	-7.275	98	.000	-7.4088000	1.0183878	-9.4297574	-5.3878426
			-7.275	97.158	.000	-7.4088000	1.0183878	-9.4299763	-5.3876237
RT UN SC	.073	.788	-4.979	98	.000	-5.2432000	1.0530211	-7.3328860	-3.1535140
			-4.979	97.440	.000	-5.2432000	1.0530211	-7.3330362	-3.1533638
F WAVE	.445	.506	-8.113	98	.000	-4.5900400	.5657731	-5.7127983	-3.4672817
			-8.113	97.539	.000	-4.5900400	.5657731	-5.7128646	-3.4672154
LTMNMC	.005	.943	-6.289	98	.000	-6.3348000	1.0072546	-8.3336639	-4.3359361
			-6.289	97.827	.000	-6.3348000	1.0072546	-8.3337081	-4.3358919
LTMNSC	2.653	.107	-3.324	98	.001	-4.7972000	1.4431364	-7.6610573	-1.9333427
			-3.324	79.672	.001	-4.7972000	1.4431364	-7.6693146	-1.9250854
F WAVE	.405	.526	-4.954	98	.000	-2.0992000	.4237081	-2.9400349	-1.2583651
			-4.954	97.421	.000	-2.0992000	.4237081	-2.9400974	-1.2583026
LT UN MC	1.617	.207	-5.641	98	.000	-5.7694000	1.0228222	-7.7991574	-3.7396426
			-5.641	94.574	.000	-5.7694000	1.0228222	-7.8000767	-3.7387233
LT UN SC	.752	.388	-5.814	98	.000	-4.65960	.80139	-6.24994	-3.06926
			-5.814	96.620	.000	-4.65960	.80139	-6.25022	-3.06898
F WAVE	4.136	.045	-6.435	98	.000	-2.8620000	.4447714	-3.7446344	-1.9793656
			-6.435	92.797	.000	-2.8620000	.4447714	-3.7452533	-1.9787467

(p value < 0.05 was considered significant) . There was no statistical significance in the goniometry between hypothyroid and euthyroid women . That is hypothyroidism does not affect the joint mobility and range of movement .

There was statistically significant difference observed between hypothyroid and euthyroid women with respect to nerve conduction study .

The motor conduction velocity showed significant difference between hypothyroid and euthyroid women bilaterally . P value was found to be less than 0.05. ($p < 0.05$) .

Similarly the sensory nerve conduction velocity also showed significant difference between hypothyroid women and euthyroid women .

There was significant difference observed in F wave response too .

Chart - 1. Showing the comparison between hypothyroid and euthyroid women in different age groups.

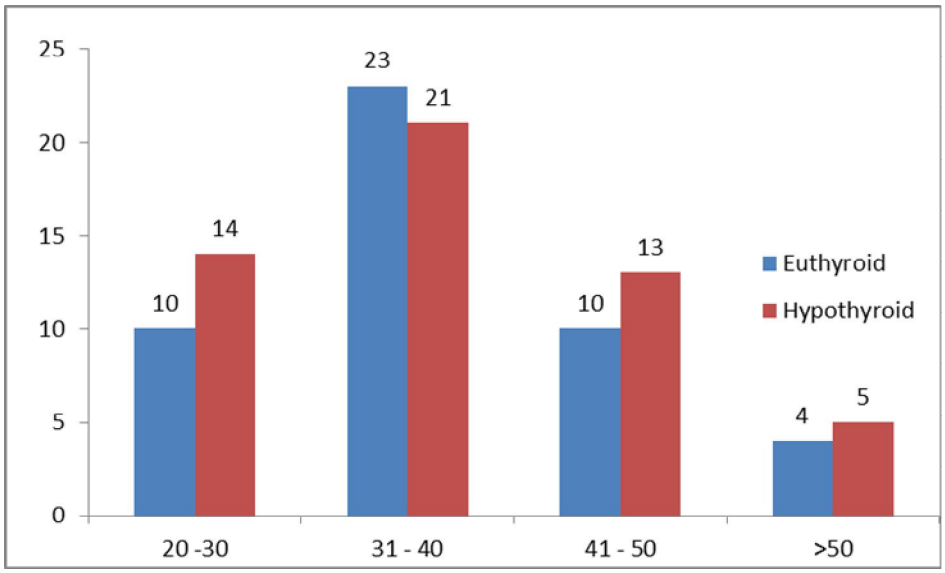


Chart – 2 Showing the comparison between Body Mass Index

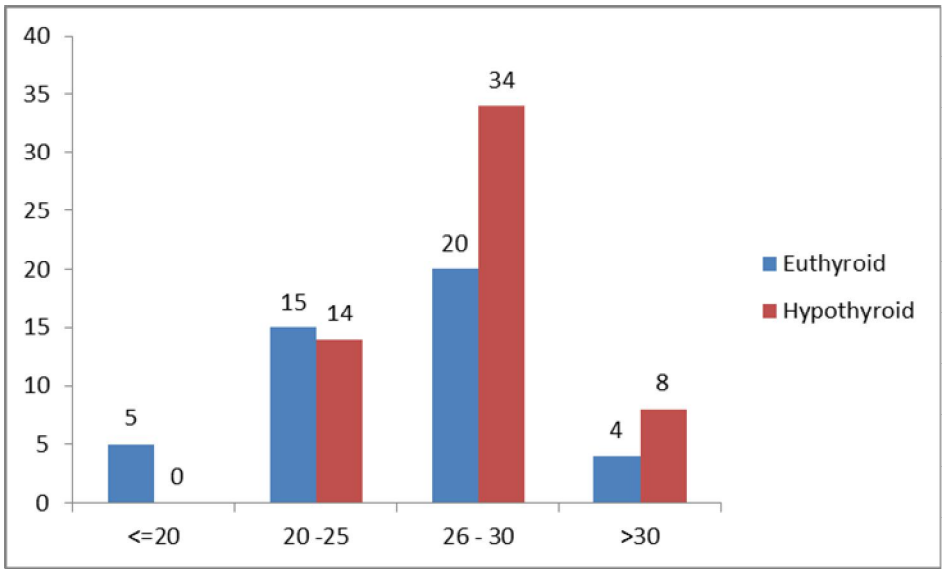


Chart - 3. Showing comparison of TSH levels between hypothyroid and euthyroid women .

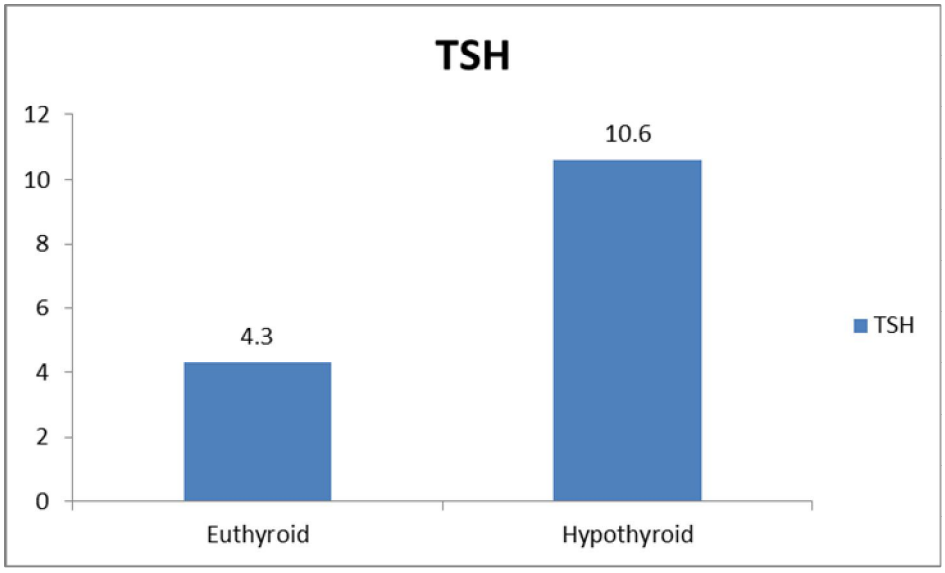


Chart - 4 . Showing Comparison of ulnar and median nerve conduction velocities

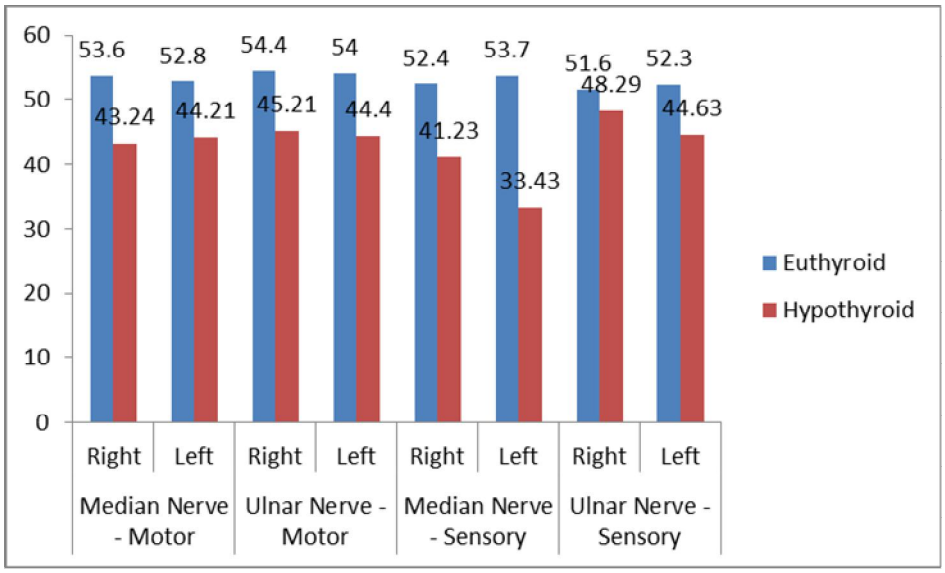


Chart-5. Showing Sensory nerve Conduction velocity

Chart-6. Showing motor nerve Conduction velocity of ulnar and median nerve

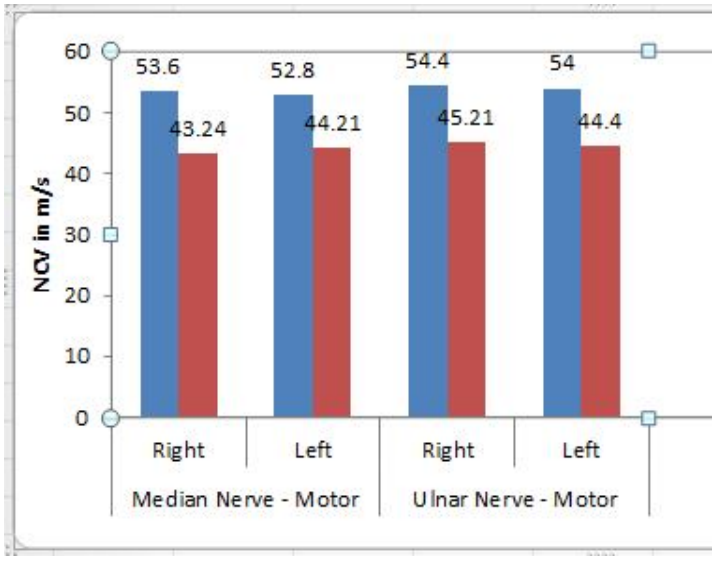
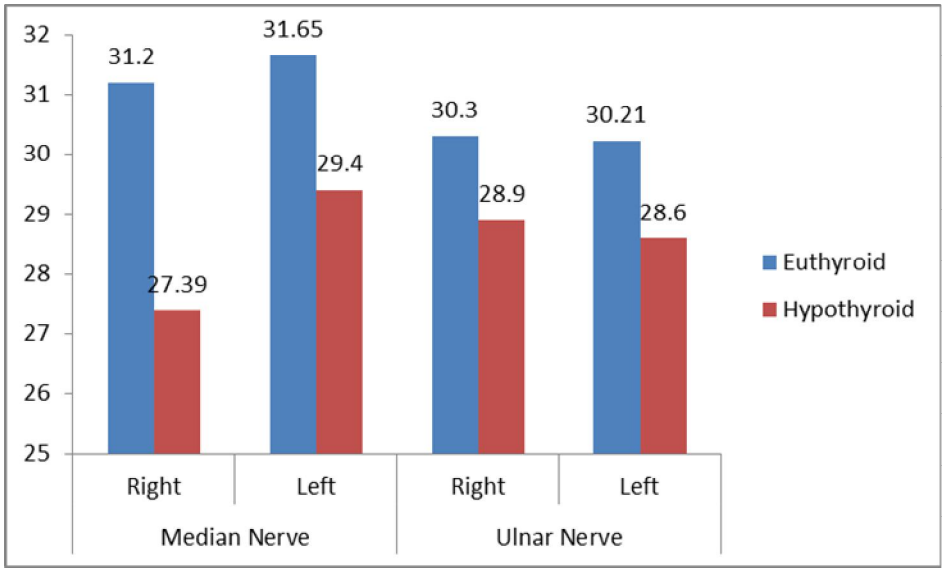
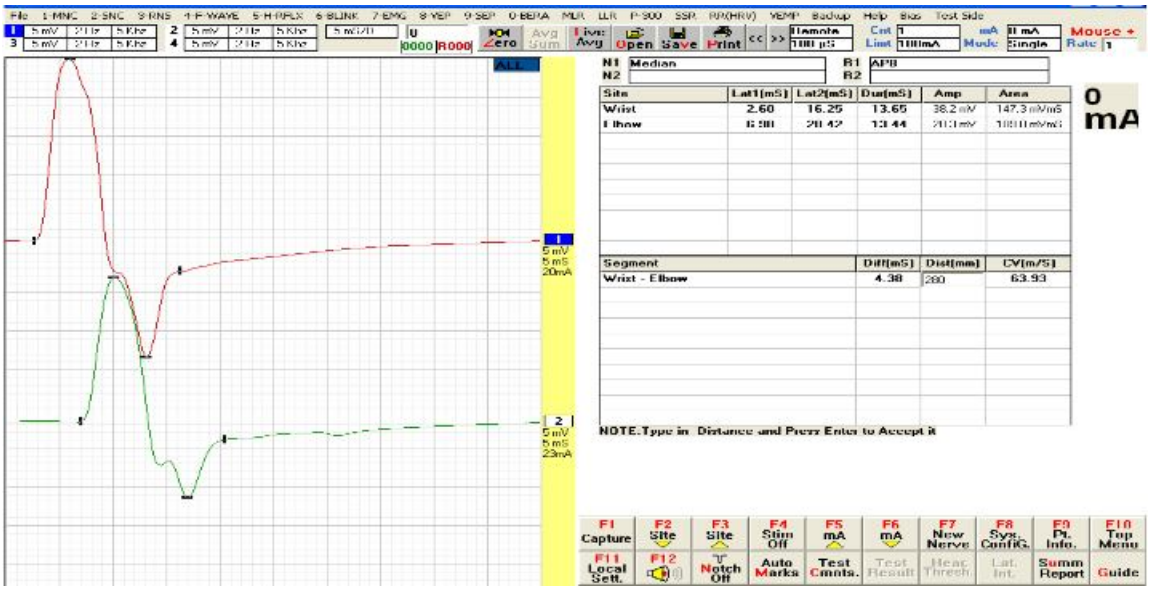


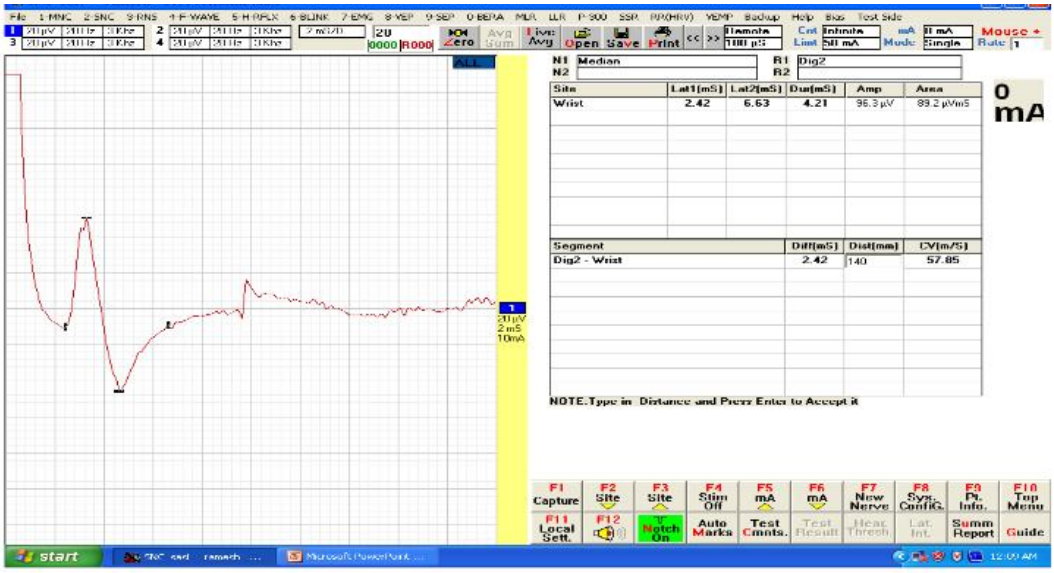
Chart -5 . Showing comparison of F wave component between ulnar and Median nerves.



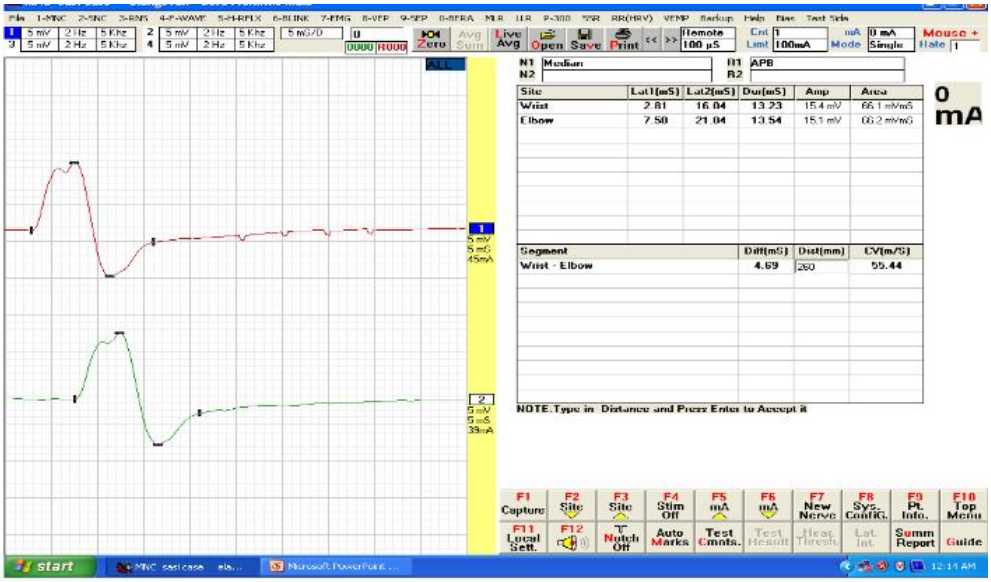
Compound Muscle Action Potential (CMAP) of Median Nerve in Normal subject



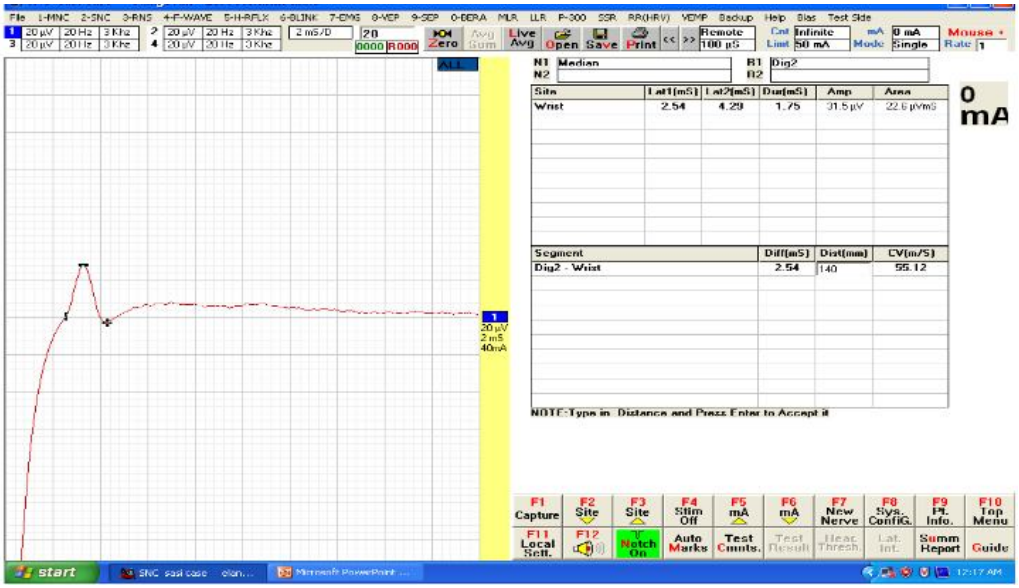
Sensory Neural Action Potential in Median Nerve in Normal subject



Compound Muscle Action Potential in Median Nerve in Hypothyroid



Sensory Nerve Action Potential in Median Nerve Hypothyroid women



DISCUSSION

DISCUSSION

Hypothyroidism is a common endocrinal disorder affecting women producing a variety of manifestations of peripheral neuropathy which affects peripheral nervous system namely motor , sensory and mixed nerves producing chronic disability .

There was correlation between the severity of neuromuscular symptoms and signs with the degree and duration of hormonal imbalance in clinical hypothyroidism (76). When motor nerve conduction velocity was still within a normal range , the sensory nerve action potentials might be reduced at an early phase of the disease. (77).

The present study showed that there was subclinical peripheral nerve involvement in newly diagnosed hypothyroid patients .

As of the goniometry recordings were concerned there was no significant change in the degree and range of movement in the joints of hypothyroid and normal euthyroid individuals . But there was significant change observed in the nerve conduction studies in hypothyroid individuals.

In my study , I had done the nerve conduction studies on the upper limb nerves , namely median and ulnar nerves . Both sensory and motor components were tested. I found that there was significant delay in sensory

nerve conduction velocity , motor nerve conduction velocity , and also delay in F wave response in the above nerves . ($p < 0.05$ was considered as significant) .

The possible causes for peripheral neuropathy in hypothyroidism could be

1. A mononeuropapthy secondary to compression caused by mucinous deposits in the soft tissues surrounding peripheral nerves and a polyneuropathy due to either a demyelinating process or primary axonal degeneration could be the possible etiology (17).

2. Nerve conduction delay in hypothyroid predominantly was due to subnormal temperature prevailing in this disorder(78).

3. A fall in thyroxine hormone has decreased membrane excitability by decreasing the sodium entry responsible for shoot up of action potential due to hyponatremia could be a possible cause. (78) .

4. Metabolic alteration in hypothyroidism affected Schwann cells leading to segmental demyelination (79).

5. Primary axonal degeneration had also been shown electro physiologically in hypothyroidism . Initially only functional loss was seen in nerve , but later structural alteration might occur as the disease progressed. (80).

6. Deposition of glycosaminoglycans , and mucin around the nerve fibers and nerve sheath leads to axonal damge (81) .

7. The pathogenesis of peripheral nerve abnormalities in thyroid dysfunction is still unclear.(17).

In the present study there was significant delay in conduction velocity in motor , sensory and F wave response unlike Adhikesavan et al (74) in whose study there was significant delay in sensory component alone among the hypothyroid women . In their study they have examined the nerve conduction velocities of left side limbs only (74) .

In the study done by Satish Waghmare et al., 100 patients of hypothyroidism, 18 years of age and above were recruited for two years from August 2012 to July 2014 . 100 age and gender matched controls were recruited for the study . Serum total T3, total T4 and TSH were determined by chemi luminescence assay in all subjects . Nerve conduction study revealed that there was significant delay in the motor nerve conduction velocity in hypothyroid individuals . The nerves examined were median , ulnar , peroneal , tibial nerves(82) .

In the study done by Jalilzadeh SH et.al., 28 individuals (25 females and 3 males) with subclinical hypothyroidism (defined biochemically as high serum TSH with simultaneously normal serum free T4) as the study group and 30 age and sex matched subjects (27 females and 3 males)

with normal thyroid function tests as the control group were enrolled in to the study . Median nerve , Ulnar nerve , sural nerve , tibial nerve and Peroneal nerve were included for Nerve conduction studies . There was no significant difference in the nerve conduction velocities between healthy controls and patients with subclinical hypothyroidism . (83) .

In another study , done by Ruchika Garg , there were forty adult females of 21 – 45 yrs of age . They were newly diagnosed cases of hypothyroids as subjects and 40 age matched women were included as controls . Neurological signs and symptoms were present in thirty five patients of hypothyroid group . The abnormal median nerve electro diagnostic findings suggestive of Carpal tunnel syndrome (CTS) due to median nerve compression at wrist was observed in 27 patients , out of which 26 were symptomatic while only one patient presented asymptomatically . They were examined before and after treatment for hypothyroidism and electrodiagnostic tests . Duration of the study was 10 months . This study revealed that hypothyroidism affects both sensory and motor components of peripheral nerves with sensory component being prominently affected (33) .

In yet another study done by Asia et al . there were 30 controls , consisting of 27 women and 3 men and 26 hypothyroid patients including 22 women and 4 men. Nerve conduction velocity studies showed that 88%

of hypothyroid patients had at least one type of electro physiological abnormality , most commonly in median and sural sensory nerves . Reduction in amplitude in 60% cases for median and sural sensory nerves and slowing of conduction velocity in 71% for these nerves was noted . There were 5 hypothyroid patients with carpal tunnel syndrome with reduction in nerve conduction velocity(84).

In the study done by Ashwini et. al., 120 women were included . There were 60 hypothyroid women and the rest 60 were euthyroid women , between 20 to 45 years of age . The nerve conduction studies revealed that the distal latency was delayed in all the nerves under consideration in hypothyroids as compared to that of control . This delay was statistically significant except for left posterior tibial nerve . The amplitude of compound muscle action potential was found to be reduced in all the nerves in hypothyroid as compared to that of control and it was statistically significant except for left posterior tibial nerve . The motor nerve conduction velocity was found to be attenuated in all the nerves of hypothyroid as compared to that of control . This slower conduction was statistically significant . (85) .

Kececi H; Degirmenci Y ., in their studies done on 40 hypothyroid individuals above 18 years of age , the nerve conduction studies had shown

that there was sensory neural conduction delay , more than motor conduction velocity(23) .

Schutt P et al . have found that hypothyroidism patients had delay in motor conduction velocity(86) .

Udayakumar et al., said that the F wave response done in median and peroneal nerve were altered in hypothyroid subjects(87) .

Sensory nerve Conduction showed an overall decrease in conduction in hypothyroid women compared to control . This decrease is well appreciated and significant in conduction velocity of sural nerve and area of both sural and median nerve . This also correlated with the study done by Ruud F Duyff et al., (76) .

Khedar et al examined 23 patients with hypothyroidism and found 52% had peripheral nervous system involvement with entrapment neuropathy in 35 % of patients (88) . The decreased conduction velocity and amplitudes were found by Lai et al., in 14 patients on peripheral nerve conduction studies (89).

Although motor nerve conduction velocity could be within a normal range sensory nerve action potentials are reduced at an early phase of the disease (77). Fincham (90) found in reduction of amplitude for median and ulnar sensory nerves .

Ozata et. al., assessed the principal electrophysiological parameters of peripheral nerves function in 27 patients with subclinical hypothyroidism and 20 age - and sex matched subjects without thyroid dysfunction. They were not able to find any significant differences between the measured electro diagnostic parameters and interpeak latencies from patient and control groups. They concluded that subclinical hypothyroidism does not lead to alterations in peripheral nerve function.(91). Only a few studies have evaluated the functional alterations in **central** and peripheral nervous systems in subjects with subclinical hypothyroidism (92) .

In the peripheral nerve conduction study done by Hande Turker et . al in Turkey , on the rats , all groups of thyroidectomized rats showed normal conduction before and after thyroxine therapy . This study indicated that , in rats : (1) the peripheral nervous system seemed to be more resistant to hypothyroidism than the central nervous system , or (2) the pathogenesis of central and peripheral nerve dysfunction in hypothyroid rats might occur through different mechanisms (20).

In the study by Ruchika Garg , mentioned earlier , both sensory and motor nerve conduction velocities were decreased with prominence of delay in sensory component , which were consistent with my results . In other studies like Schutt P et al ., Ashwini A et al , and Satish Waghmare et al. there was significant delay in motor conduction velocity of the examined nerves .

Ruud F Duyff et al., Kececi H ; Degirmenci Y ., Asia et al., Adhikesavan et al . had found that sensory nerve conduction velocity was delayed .

Udayakumar et al ., had found that there was significant delay in F wave response .

Jalilzadeh SH et al., had found no delay in both sensory and motor nerve conduction velocities in hypothyroid women .

This involvement of both sensory and motor components of peripheral nerves could be due to subclinical inflammation of the nerves in hypothyroidism leading to release of free radicals causing axonal degeneration and subsequently demyelination of the peripheral nerves (74) .

SUMMARY

SUMMARY

A study was conducted to evaluate the peripheral nerve conduction through nerve conduction studies and to assess the degree and range of movement of joints of the limbs by using the goniometer in hypothyroid and euthyroid women .

After obtaining the institutional ethical committee approval , and appropriate consent from the individuals , the nerve conduction study and goniometry was done on hypothyroid women and age matched euthyroid women . 50 hypothyroid women and 50 euthyroid women were recruited for this study . They were selected from female medical out patient department of Coimbatore medical college hospital , Coimbatore . This study was done between July 2015 and March 2016 .

Biochemical assessment was done in clinical biochemistry laboratory , using ELISA technique to evaluate the thyroid hormone and TSH levels . Anthropometric measurements like height , weight , body mass index were done at non communicable disease out patient department, at Coimbatore medical college hospital, Coimbatore. Nerve conduction study and Goniometric measurements were done at neurology laboratory, Coimbatore medical college hospital, Coimbatore.

The data collected from the above study was analysed statistically, using the SPSS software version 16, and Independent ‘t’ test.

Goniometric measurements of the limb joints of hypothyroid women and euthyroid women did not reveal any difference. These measurements remained within normal range. But the nerve conduction studies done on hypothyroid women revealed that there was significant delay in motor and sensory component of peripheral nerves (median , and ulnar nerves). Some hypothyroid patients also showed delay in F wave component. p-value less than 0.05 was considered significant.

Thus this study concludes that the hypothyroid women have delay in peripheral nerve conduction velocities, including sensory and motor components and the degree and range of movements of limb joints were not affected. Nerve conduction study may be utilized as a routine screening test for hypothyroid individuals, so that the hormone replacement therapy can be instituted at the earliest .

CONCLUSION

CONCLUSION

The following conclusions have been derived from the present study :

It is proved from this study that early hypothyroidism causes subclinical neuropathy as significant variations were found in almost all the nerve conduction parameters when compared to normal individuals.

It is characterized by predominant and early involvement of sensory fibers.

It is well established that thyroid hormones have profound effects on mitochondrial oxidative activity, synthesis and degradation of proteins , sensitivity to catechol amines , differentiation of muscle fibers , capillary growth and level of antioxidant enzymes and compounds (93).

Deficiency of thyroid hormone leads to increase in adipose tissue deposition, which in turn leads to subclinical inflammation , causing free radicals release that damages the nerves . (Demyelination and axonal degeneration) . Axonal degeneration is the primary pathology which is associated with secondary demyelination (94) .

Nerve conduction study is a simple and noninvasive procedure which should be done as a routine investigation in hypothyroid patients to pick up early changes occurring in the nerves since the prognosis of mild and moderate neuropathy is good . Degree and range of movements of the joints (ROM) measured by Goniometry has no relavence to hypothyroidism .

LIMITATION OF THIS STYDY

1. In this study only women were included. Men were not included .
2. The nerve conduction study was done on only upper limb nerves, namely ulnar and median nerves.
3. This study was done on hypothyroid women who had not been started on thyroxine. Response of Post thyroxine therapy was not assessed.

FUTURE SCOPE OF THIS STUDY

Nerve conduction study can be used as an effective tool for early diagnosis of subclinical hypothyroidism and associated peripheral neuropathy.

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ANNEXURES

MASTER CHART

MASTER CHART : NERVE CONDUCTION READINGS AND GONIOMETRY MEASUREMENTS

sl.no	Name	Age yrs	Ht	Wt	BMI	T3	T4	TSH	1 OR 2	RT SHOULD	LT.SHOULD	RT.ELBOW	LT.ELBOW	RT WRIST	LT WRIST	RT MCP	LT MCP	RTMNMC	RTMN SC	F WAVE	RTUNMC	RT UN SC	F WAVE	LTMNMC	LTMNSC	F WAVE	LT UN MC	LT UN SC	F WAVE
1	SIRAJ	47	1.62 MTS	70 KGS	28	0.94	11.8	1.25	2	55	55	70	70	15	15	60	60	58.55m/s	50.42	28.3	58.95	52.08	30.6	61.58	48.78	30	52.14	52.08	30.8
2	FARIDHA	42	1.65	69	26	8	11.4	0.83	2	55	55	55	55	30	30	84	84	58.08	50.42	27.4	64.9	51.02	34.84	60.11	48	29.5	58.95	50	31.8
3	GIRIJA	60	1.58	61	24	1.18	11.8	1.1	2	35	35	50	50	40	40	65	65	54.78	43.78	32.3	76	52.23	36	54.11	53.21	32.11	53.02	49.23	32.23
4	SUMADY	46	1.51	57	24	1.89	9.6	0.09	2	55	55	60	60	40	40	74	74	58.67	46.51	32.2	62.18	53.19	28.9	60.27	55.3	29.3	60.61	55.87	28.4
5	DEEPA	22	1.52	58	25	0.68	5.02	13.23	1	55	55	65	65	30	30	70	70	43.24	41.23	25.23	45.21	43.32	22.22	49.23	44.21	24.45	44.21	40.11	21.23
6	JUWARIA	21	1.58	61	24	0.88	9.1	3.06	2	55	55	75	75	45	50	20	35	54.95	42.64	27.3	61.97	24.52	31.8	50.22	52.88	30	55.8	45	32.7
7	SAFIYA	25	1.59	74.5	28	1.07	7.8	2.4	2	70	70	80	80	40	60	60	60	63.01	49.59	29.9	68.49	53.19	31.5	63.13	54.3	25.2	63.01	61.73	30.4
8	VALLI	23	1.54	57	24	0.45	0.05	10.4	1	70	70	80	80	60	60	40	40	43.25	43.43	25.12	46.32	51.12	32.12	34.53	56.22	32.43	55.23	54.32	32
9	SAHAYA M	47	1.48	41	18	1.1	7.9	0.3	2	60	60	70	70	50	50	70	70	53.21	50.23	34.21	57.32	53.15	31.32	63.23	51.23	34.36	56.39	56.28	34.33
10	SIVAGAMI	26	1.45	51	24	0.64	6.7	7.8	2	70	70	80	80	50	50	50	50	59.56	57.34	37.34	58.34	58.34	38.23	56.49	49.39	35.34	53.49	53.21	36.34
11	VALARMAT	35	1.52	72	31	0.9	4.3	11.93	1	80	80	135	135	40	40	67	67	44.15	37.38	32.3	60.1	59.88	27.2	30.67	0	32.7	59.95	58.48	30.5
12	JANNATH	21	1.54	70	29	0.23	4	10.4	1	75	75	70	70	45	45	25	25	58.67	51.89	25.1	64	55.56	24.6	63.95	53.92	25.5	67.8	50.28	24.1
13	JAYANTHI	34	1.44	72	34	0.12	2.1	11.4	1	50	50	55	55	35	35	35	35	56.65	37.85	31.4	68.49	57.14	30	56.65	44.94	30.2	66.67	57.14	31.3
14	PADMAVA	32	1.55	60	25	1.4	3.5	9.77	2	65	65	75	75	45	45	47	47	36.8	0	34.6	55.8	52.63	26.2	42.51	33.43	28.1	59.95	53.89	26.9
15	AMUDHA	34	1.61	64	25	0.5	5.2	7.89	1	40	55	70	75	40	50	77	80	51.34	48.34	32.63	48.23	51.21	31.29	46.21	32.15	29.1	50.21	51.29	29.32
16	VASANTHA	38	1.55	71	30	0.82	4.2	15.38	1	30	34	65	73	38	30	70	77	49.29	48.39	31.2	49.34	48.49	30.78	45.29	34.39	31.27	44.63	50	30.23
17	NASIMA	47	1.47	40	19	0.97	10.4	3.3	2	45	55	98	100	28	28	84	84	54.19	52.34	32.34	56.23	52.19	34.65	53.32	53.23	31.56	63.28	54.38	34.37
18	TAMILSE	38	1.6	57	22	0.56	4.3	18.2	1	55	55	75	80	40	40	65	60	50.45	49.37	30.38	45.34	54.38	32.3	50.32	48	32.34	48.37	51.43	31.24
19	NAGAMAN	39	1.52	64	28	0.99	11.4	0.93	2	60	65	80	80	50	45	60	55	58.23	56.78	35.22	65.23	58.45	37.54	69.21	59.23	38	53.21	58.56	39.23
20	SALEEMA	50	1.51	70	30	1.23	13.1	2.74	2	35	30	60	50	30	30	55	50	55.21	56.34	32.24	64.43	58.32	38.32	61	54.25	34.35	54.47	57.38	38.23
21	SELVI	34	1.49	64	28	0.42	4.72	TOO HIGH	1	55	55	65	65	50	50	55	55	42.1	48.4	30.21	50	51.21	30	42.45	44.5	25.21	43.15	49.21	28.35
22	BAGAVATH	26	1.48	59	27	0.67	3.8	16.5	1	60	60	70	70	35	35	40	45	49.39	43.23	30.11	51.4	45.47	29.4	49.13	50.14	30.34	44.49	48.32	32.1
23	KARPAGAN	45	1.6	65	25	1.2	13.67	0.2	2	65	70	70	65	50	55	70	74	58.32	54.65	38.32	57.22	60	35.88	60.21	55.2	35.23	56.53	57.32	35.44
24	DEEPA	25	1.55	70	31	0.84	3.2	11.36	1	65	60	60	60	65	59	70	75	48.47	49.32	28.43	49.2	49.22	29.4	53.21	52.21	30.21	45.32	47.45	29.54
25	KAMALA	28	1.41	64	32	0.44	2.8	17.5	1	55	50	50	60	60	55	65	65	47.54	44.23	29.04	43.21	33.23	32.54	53.87	39.39	29.78	43.64	44.54	30.21
26	NOORUN	32	1.58	70	28	0.9	4.9	9.3	2	70	70	65	65	60	60	55	50	53.32	57.54	38.39	54.35	40.38	32.7	54.32	40.56	32.65	57.34	45.65	39.54
27	ANBUSELV	35	1.54	70	29	0.5	4.7	8.9	2	65	65	70	70	60	60	55	55	56.34	57.75	31.32	54.45	56.46	34.6	57.36	45.39	33.25	63.21	44.94	32.29
28	MAHALAXI	54	1.58	61	24	0.9	6.4	9	2	60	63	75	75	67	65	60	60	55.34	58.32	32.33	58.34	54.3	33.65	51.25	53.23	34.65	53.78	56.32	31.53
29	ZAINAB	38	1.56	78	32	0.4	2.3	18.2	1	60	60	65	65	60	60	56	55	46.5	43.2	28.2	44.82	32.65	28.9	42.25	50	30.34	45.43	45.76	29.7
30	ARULSELVI	32	1.57	45	18	1.1	8.2	7.2	2	56	55	65	65	75	75	60	60	53.2	49.32	32.32	52.6	56.56	32.4	51.75	53.26	31.56	54.32	54.23	31.64
31	DEEPA	47	1.49	63	28	0.4	4.2	12.3	1	70	70	65	65	60	60	65	65	47.26	47.44	30.65	44.65	47.3	29.3	43.21	45.28	30.36	46.37	42	30.21
32	PAPPATHY	53	1.54	63	26	7.79	10.07	0.8	2	65	65	75	75	70	70	50	50	53.23	54.76	32.56	57.75	50.56	34.37	54.64	53.23	38.3	47.32	54.29	34.54
33	SOWMYA	30	1.56	70	29	0.32	3.1	13.3	1	56	57	50	60	70	70	65	65	40.45	50.1	30.65	45.64	41.44	32.65	46.5	42.3	32.21	43.9	46.32	31.93
34	ANITHA	37	1.58	77	31	0.34	2.6	15.32	1	70	70	65	65	70	70	50	50	49.32	47.75	29.15	45.17	43.29	29.46	47.48	43.26	32.78	44.37	48	31
35	DIVYA	26	1.55	71	30	0.23	3.2	14.2	1	75	75	50	50	55	55	60	60	43.76	42.75	29.39	45.27	41.58	30	46.32	40.57	31.75	39	40.83	30.67
36	RANI	42	1.44	67	32	0.35	3.7	13.9	1	60	60	65	65	70	70	65	65	44.15	43.78	31.2	43.23	43.26	30.32	45.56	47	28.76	45.9	47.43	33.67
37	SAKTI	37	1.6	65	25	0.9	5.8	8.3	2	57	57	80	80	65	65	70	70	59.38	53.65	34.56	55.32	53.9	33.76	56.03	54.74	31.65	56.45	50.45	34.74
38	PRIYA	45	1.54	57	24	1.5	6.3	6.7	2	50	50	70	70	75	75	65	65	54.38	58.9	32.75	57.87	53.74	32.89	58.34	53.32	34.32	53.26	57.83	36.5
39	INDIRA	50	1.56	63	26	2.3	7.4	9.3	2	76	75	70	70	65	65	50	50	58.32	54.34	32.53	58.37	49.33	37.352	58.36	55.37	31.18	58.78	59	30.87
40	RADHA	34	1.45	54	25	0.37	2.3	15.3	1	57	60	65	65	70	70	73	75	45.2	42.6	31.2	43.8	41	30.3	45.65	33.48	36.32	43.76	41.39	30.32
41	MALLIGA	43	1.52	64	28	1.78	6.3	8.9	2	70	70	56	55	78	78	80	80	53.57	58.32	35.75	55.39	54.76	39.23	52.1	38.2	32.45	53.67	52.48	32
42	RAZIA	22	1.56	64	26	1.38	7.3	8.3	2	75	75	80	75	55	55	65	65	54.32	56.47	33.2	59.3	53.87	38.67	51.24	43.3	33.65	35.32	53.21	34.53
43	GOWRI	43	1.67	70	25	1.23	5.7	10.2	2	75	75	65	65	55	55	70	70	53.78	55.37	39.2	54.47	52	34.54	54.38	53.9	33.74	49.3	52.1	35.48
44	KOSALYA	57	1.58	65	26	2.6	6.8	7.3	2	60	60	57	58	65	65	70	70	52.32	54.89	31.38	55	55.93	39.4	52.81	54.76	32.67	52.87	56.3	32.3
45	ARTHY	32	1.54	63	26	2.1	3.2	3.3	1	75	75	65	65	60	60	70	70	51.2	52.5	32.39	47	47.8	39	50.2	53.7	29.87	49.9	52.3	30.56
46	KALPANA	34	1.48	59	27	2.8	4.7	4.3	2	55	55	70	70	60	60	75	75	49.2	51.6	31.4	39	46.3	37.33	50.3	46.5	32.23	50.2	51.6	29.5
47	GAYATHRI	35	1.58	74.5	28	1.2	2.2	11.5	1	60	60	74	74	65	65	75	75	43.2	47.5	34	42.6	48.3	33.32	49.3	44.3	29.37	40.5	44.2	29.46
48	VIJI	48	1.52	58	25	1.5	2.3	2.59	2	55	55	70	70	65	65	60	60	49.2	44.3	30.45	50.3	49	38.45	39.65	42.43	30.43	43.34	44.64	30.35
49	ZUBAIDA	36	1.52	64	28	1.5	2.5	7.6	1	65	65	55	55	75	75	56	56	50.2	51.3	34.83	44.3	50.74	39.74	42.3	49.49	31.45	52.3	47.3	32.2
50	KAVIYA	42	1.54	70	29	0.56	1.34	15.32	1	65	65	75	73	75	75	65	65	52.1	51.2	30.63	50.83	51.63	37.32	43.34	50.76	32.32	42.33	53.2	32.21
51	AALIYA	34	1.53	62	26	1.1	3.2	4.4	2	65	65	56	56	75	75	60	60	53.2	51.4	33.83	52.64	57.24	35.52	50.63	54.42	34.23	49.67	50.21	35.74
52	TAENMOZI	36	1.49	63	28	0.32	1.45	16.3	1	65	65	70	70	65	65	75	75	50.21	49.32	29.25	43.92	41.42	30.21	47.21	43.64	31.26	40.37	48.38	29.17
53	DEVI	47	1.52	57	24	1.4	3.2	3.1	1	70	70	65	65	75	75	55	55	52.83	53.2	33.2	51.53	56.34	34.3	54.67	55.3	35.25	53.65	57.32	32.37
54	KAVITHA	40	1.6	74	28	1.83	2.31	9.3	1	55	55	70	70	65	65	70	70	49.22	50.23	31.32	47.64	50.65	30.64	52.65	49.64	31.63	49.38	52.35	29.65
55	LEENA	45	1.58	67	27	1.12	3.5	8.3	1	50	50	55	55	65	65	70	70	50.28	51.37	32.2	53.11	49	31.42	51.31	49.28	30.21	50.21	51.45	31.4
56	HELEN	40	1.64	70	26	0.92	2.3	2.4	2	70	70	55	55	65	65	75	75	53.23	55.4	33.67	49.67	51.45	32.45	52.31	50.25	32.41	53.12	58.2	33.53
57	SONA	38	1.48	53	24	1.02	2.7	1.45	2	57	55	74	75	70	70	65	65	53.32	52.35	32.32	53.89	54.52	34.53	51.32	50.35	34.32	55.35	57.34	30.32
58	VAISALI	41	1.45	52	25	1.1	2.3	1.74	2	50	50	75	75	60	60	75	74	55.3	54.32	32.32	53.45	55.56	32.53	49.36	57.32	33.57	57.89	53.57	31.36
59	ARCHANA	32	1.58	65	26	1.3	1.38	2.1	2	75	75	65	65	70	70	75	75	53.25	56.34	32.32	57.32	52.67	34.53	53.63	51.65	31.53	52.45	51.64	31.4
60	HASINA	35	1.63	74	28	0.42	1.34	13.4	1	75	75	60	60	65	65	80	82	44.93	49.38	30.21	49.56	48.76	29.83	51.2	49.73	30.34	48.84	50	28.92
61	PUVITHA	29	1.49	57	26	1.02	2.1	2.3	2	55	55	80	80	70	70	65	65	52.34	55.35	32.53	53.64	50.21	31.5	57.32	51.2	32.76	58.34	53.1	33.65
62	ANJALAI	34	1.7	77	27	0.37	1.4	16.3	1	60	60	75	75	77	78	55	55	50.35	50.83	31.3	51.3	49.73	29.3	52.63	48.32	30.87	52.75	49.73	30.65
63	JAYANTHI	44	1.64	84	31	0.49	0.67	15.7	1	60	60	75	75	70	70	65	65	44.65	45.67	28.56	48.76	46.73	30.65	47.76	49.3	31.31	42.36	50.34	27.73
64	SUGANTHY	32	1.53	62	26	0.44	1.3	14.9	1	65	65	77	75	65	65	70	70	43.83	48.39	31.7	49.59	48.49	29.39	44.98	41.3	29.83	50	41.35	30.76
65	YAZINI	43	1.63	70	26	1.37	2.39	3.87	2	60	60	56	55	75	75	65	65	52.37	55.64	32.43	58.78	57.32	34.7	58.46	56.45	32.28	56.32	50.63	34.45
66	SAROJA	29	1.67	70	25	1.05	1.87	4.2	1	75	75	70	70	65	65	58	60	50.04	50.8	29.3	48.83	44.76	29.4	51.4	49.74	30.33	48.43	46.74	28.45
67	LEELA	36	1.49	63	28	0.73	1.23	3.45	1	68	73	70	70	65	65	70	70	51.48	50.38	30.5	51.9	48.56	28.54	50.21	51.34	31.32	45.37	49.36	29.34
68	KAMALAM	44	1.56	66	27	0.43	0.83	12.89	1	75	75	60	60	65	65	75	75	49.3	49.8	29.6	48.3	47.65	27.45	49.8	50.5	29.7	47.74	51.3	29.3
69	AADHILAXI	41	1.6	74	29	0.54	0.95	13.43	1	70	70	60	60	65	68	70	70	50.8	51.7	28.3	46.4	49.36	29.73	50.74	52.86	30.3	49.65	44.36	29.37

70	GEETHA	28	1.67	72.3	25	1.35	3.5	4.2	2	75	75	70	70	80	80	65	65	52.21	53.6	30.5	55.83	57.63	34.2	56	51.3	31.35	55.8	52.39	32.64
71	NITHYA	37	1.59	55	22	0.73	2.4	2.8	2	74	75	70	70	65	65	75	75	53.78	54.65	32.5	54.63	56.73	36.35	51.45	55.67	33.5	54.65	51.46	32.45
72	SURABI	33	1.65	68	25	0.74	0.97	2.5	2	77	75	70	70	65	65	75	75	58.43	56.65	29.56	57.46	56.4	37.58	53.8	52.65	32.7	55.36	56.3	31.5
73	INDIRA	40	1.58	62	25	0.75	2.4	3.4	2	73	75	70	70	65	65	70	70	52.5	57.3	30.4	53.76	56.36	34.75	56.75	52.67	32.53	56	54.35	33.54
74	BANU	36	1.52	70	30	0.32	0.57	13.2	1	60	60	75	75	60	60	65	65	50.2	49.4	28.3	51.3	50.6	30.34	52.8	55.65	29.54	49.74	47.4	31.25
75	RADHA	32	1.51	55	24	0.92	1.32	2.3	2	80	80	67	65	75	75	65	65	54.38	55.94	31.5	56.3	56.54	33.89	57.94	53.28	32.43	53.76	56.74	33.32
76	DANAM	43	1.48	64	29	0.34	0.75	13.5	1	55	55	65	65	70	70	75	78	50.35	50.24	30.4	51.73	50.15	28.45	48.54	51.47	30.73	50.28	49.47	30.54
77	INDHUMAI	28	1.55	74	31	0.54	0.84	12.93	1	65	65	75	78	70	70	60	60	49.38	50.2	28.73	51.2	48.73	29.43	51.32	52.38	30.36	51.23	53.12	29.38
78	RAJI	34	1.57	75	30	0.39	1.2	9.75	1	60	60	65	63	70	70	63	65	50.37	51.54	29.78	52.39	49.83	28.47	50.29	51.27	30.3	50.73	51.39	29.83
79	ILAVARASI	38	1.54	60	25	1.2	2.3	2.4	2	70	70	60	60	65	65	70	70	54.3	56.45	32.32	53.38	58.37	34.56	53.74	57.32	32.73	54.75	56.3	32.37
80	DURGA	33	1.6	73.5	29	1.4	0.89	3.2	2	70	72	65	65	75	75	60	62	53.36	57.35	30.1	50.23	54.74	35.42	55.73	57.36	33.7	56.45	53.63	31.19
81	USHA	28	1.54	70	29	0.76	1.3	2.6	1	70	70	65	65	75	75	60	60	50.65	50.56	29.5	49.67	50.1	29.76	51.8	50.5	30.45	50.87	50.47	29.65
82	KALYANI	30	1.49	63	28	1.3	1.6	2.8	2	70	70	60	60	65	65	67	65	55.45	56.46	30.7	57.54	53.28	36.56	54.65	54.85	29.4	54.29	52.59	30.43
83	JULIA	30	1.57	69	28	0.44	0.87	2.3	1	76	75	60	60	56	55	60	60	51.22	50.38	30.24	49.35	52.38	29.65	51.33	48.58	29.9	49.76	51.59	30.3
84	RANI	41	1.53	70	30	0.37	2.2	11.3	1	80	82	70	70	65	65	55	55	51.43	50.37	29.3	52.5	50.76	29.48	51.35	52.53	30.19	48.67	52.45	31.3
85	CATHRIN	38	1.56	73	30	0.39	0.67	10.83	1	75	75	80	80	65	65	70	70	48.38	50.67	30.23	49.65	52.37	28.36	49.65	50.37	28.38	52.73	50.73	29.39
86	SATHYA	33	1.54	73	31	0.33	0.65	14.35	1	55	55	66	65	70	70	60	60	44.35	48.38	28.3	47.39	50.35	28.47	49.73	51.38	28.11	50.18	46.43	28.44
87	MINI	40	1.61	82	32	0.22	1.2	13.9	1	50	50	60	60	55	55	65	65	50.25	49.26	29.4	45.81	49.84	29.47	51.23	52.58	28.35	51.21	47.61	30.1
88	VANI	29	1.6	63	24	2.4	3.2	2.4	2	60	60	65	65	75	75	70	70	56.32	55.18	32.13	53.19	57.41	36.54	58.35	53.83	32.13	56.73	57.31	32.36
89	NAYAGI	31	1.58	73	29	0.32	0.89	8.3	1	60	60	65	65	70	70	55	55	50.32	51.23	29.3	49.53	48.5	28.76	50.38	52.18	30.32	49.11	44.19	29.8
90	MARY	32	1.49	68	31	0.48	0.83	7.53	1	70	70	56	55	75	75	65	65	50.65	52.81	30.3	49.19	48.18	28.45	50.32	55.3	29.39	51.23	52.28	30.3
91	ANANDI	31	1.59	65	26	2.1	1.8	2.9	2	60	60	75	75	65	65	55	55	52.3	55.16	32.14	53.78	56.91	36.5	52.34	54.79	32.19	53.75	55.39	33.17
92	YAMUNA	28	1.57	70	28	1.9	2.2	2.7	2	65	65	55	55	75	75	60	60	54.19	55.17	30.17	55.71	56.81	37.94	56.12	54.11	31.18	54.82	53.39	32.18
93	AARTHY	38	1.54	70	29	0.41	0.84	12.81	1	55	55	65	65	75	75	70	70	50.21	49.13	29.3	52.31	50.32	28.9	51.25	50.17	29.78	49.29	54.74	30.18
94	DEIVANAI	31	1.59	71	28	0.45	1.3	10.1	1	58	55	65	65	75	75	70	70	51.19	52.18	30.14	51.17	50.1	27.43	51.71	49.31	29.31	51.44	54.18	31.17
95	CHANDRA3	23	1.55	60	25	2.1	3.5	2.1	2	70	70	75	75	65	65	75	75	54.39	57.41	32.15	53.81	56.83	36.4	55.31	57.42	32.21	53.83	57.49	31.34
96	MANGAI	32	1.63	65	24	1.8	2.9	2.7	2	70	70	75	75	65	65	60	60	53.31	57.32	31.2	54.91	56.01	32.35	58.31	59.36	32.13	54.32	56.39	32.3
97	VALLI	30	1.6	75	29	0.31	1	2.8	1	65	65	75	75	60	60	85	80	52.31	51.58	30.16	49.16	50.62	29.95	50.14	52.3	29.81	52.21	50.15	29.17
98	KAVITHA	32	1.56	60	25	1.8	1.58	3.1	2	70	70	65	65	75	75	70	70	55.32	58.32	33.2	57.03	54.93	33.74	56.31	54.36	32.83	55.81	58.31	32.07
99	KAAVIYA	33	1.5	60	27	1.3	2.1	3.3	2	60	60	56	55	75	75	65	65	55.31	52.14	32.01	56.95	54.3	32.74	59.3	54.73	32.31	53.38	53.08	31.73
100	MAGESVAI	29	1.59	65	26	2.1	3.1	2.8	2	75	75	70	70	56	55	65	65	53.3	53.08	32.83	58.31	57.04	37.49	53.71	55.03	31.09	56.48	55.38	33.04

H = Hight Wt=Weight BMI= Body Mass Index T3 = Tri iodo Thyronine T4= Thyroxine TSH = Thyroid Stimulating Hormone 1 = Hypothyroid individual 2 = Euthyroid individual

RT = Right LT = Left MCP = Meta Carpo Phalangeal Joint Rt MN MC =Right Median Nerve Motor Conduction LT MN MC =Left Median Nerve Motor Nerve Conduction SC = Sensory Nerve Conduction UN = Ulnar Nerve

PROFORMA

1. Name:

2. Age:

3. Address:

4. Your Phone No:

5. Do you have Thyroid disorder:

6. Since how long you suffer from this disorder:

7. Any of your family members suffer from thyroid disorder:

8. Since how long do they suffer from this disorder :

9. Have you taken medicines for thyroid disorder earlier :

10. For how long you have taken those medicines

11. Do you suffer from Hypertension :

12. Since how long you suffer from hypertension :

13. What is your blood pressure at present:

14. Do you have Diabetes mellitus :

15. For how long do you suffer from diabetes :

16. Do you have any musculo skeletal problems :

17. For how long you suffer from that :

18. Do you have any neurological abnormalities :

19. For how long you suffer from that :

20. How much is your Height :

21. How much is your weight :

22. How much is your BMI :

23. What is your present T3 level :

24. How much is your T4 level :

25. How much is your TSH level:

26. How much is your blood glucose level :

CONSENT FORM

I am…….. D/O W/O Mr…… resident of ……

…….suffering from thyroid disorder (Hypothyroidism). I have been explained about the consequences of hypothyroidism including neurological problems. Hence I am willing to undergo the Nerve conduction study done by Dr. K.S. Madhar Shah, a post graduate student of Physiology Department of Coimbatore Medical College, Coimbatore. The procedure of this study has been explained to me, and I am aware of withdrawing myself from this study at any time. I am not forced by anyone for this testing.

Station :

Date : Signature of the individual

ஒப்புதல் / இசைவுப்படிவம்

நான் தருமதி / செல்வி .....

க.பெ / த. பெ. .....

விலாசம் .....

தைராய்டு சுரப்பி குறைவு நோயினால் பாதிக்கப்பட்டுள்ளேன். இதனால் என் நரம்பு மண்டலம் பாதிப்புக்கு உள்ளாகலாம்.

எனவே கோவை மருத்துவக் கல்லூரி உடலியங்கியல் துறையில் மருத்துவ பட்ட மேற்படிப்பு மாணவரான மரு.சையத் மதார் ஷா மேற் கொள்ளும் நரம்பு கடத்து திறன் சோதனைக்கு உட்பட நான் முழு மனதுடன் சம்மதிக்கிறேன். இந்த சோதனையின் பின் விளைவுகள் எனக்கு தெளிவாக விளக்கப்பட்டுள்ளது.

இந்த சோதனையில் ஈடுபட நான் மருத்துவரினால் கட்டாய படுத்தப்படவில்லை என்றும் எந்த நேரத்திலும் இந்த சோதனையில் இருந்து விடுபட எனக்கு முழு உரிமை உண்டு என்பதையும் நான் நன்கு அறிவேன்.

மருத்துவர் கையொப்பம்:

தேதி : கையொப்பம்